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INFECTION WITH LOA LOA

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Having observed 2 cases of infection with Loa loa, we are prompted to epitomize some of the available information regarding this disease. It is exceedingly rare in the United States but is endemic on the west central coast of Africa, being particularly prevalent in the Cameroons. Sharp [1] reported that in the Cameroons, with a population of 63,000 in 6,000 square miles, 6 per cent of the population were affected. We gained the impression from both our patients that the incidence of the disease is much greater. They ventured the opinion that nearly all of the natives and a good proportion of the white population are victims of filariasis due to L. loa. The disease has been known for over three hundred years. Both our patients were missionaries returned from Africa. Their histories were parallel, and we report here the most interesting one.

REPORT OF A CASE

Miss E. M., aged 37, a missionary from French Cameroons, arrived in Africa on April 1, 1932. She soon heard other missionaries speak of aches and swellings near articulations, and of so-called "eye worms" that wriggled across the eye. She learned that practically the entire population was infected with L. loa.

Her medical history was irrelevant, and her general physical condition was excellent. She stated that a fly, called by the natives the "osun," would bite repeatedly while she was seated at an organ, listening to a sermon or otherwise engaged than in continuously protecting herself. During her second year of residence in the tropics a microfilaria was discovered by accident in a blood smear stained for malaria, but no clinical symptoms appeared until 1936, approximately two years later. Swelling of the ankles was first noted. This was so severe that elephantiasis was suspected. The swellings spontaneously disappeared after a time, and several months later localized, slightly inflamed swellings, associated with aching and burning, appeared on the arms. The individual lesions would last about two days and then spontaneously disappear, others making their appearance from time to time. About the same time a worm appeared under the bulbar conjunctiva of the right eye, where it was easily visible. Considerable swelling and pain were present. Ice compresses caused disappearance of the worm after forty-eight hours (there was no physician available for other treatment). The "Calabar swellings" appeared at intervals on the arms, and at times the hands were swollen to the extent that closing them was impossible. Lesions located about the wrists were more severe than others, being at times swollen and hot and often marked by wheals in the form of irregular red lines or circles (fig. 1).

In October 1940, after the patient had returned to the United States, a worm appeared beneath the bulbar conjunctiva of the left eye. Some swelling and pain were noted, but the symptoms were less severe than those experienced in Africa with the first worm. Dr. Paul

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1. Sharp, N. A. D.: Filariasis in the Cameroon, Tr. Roy. Soc. Trop. Med. & Hyg. **21**:413-416 (Feb.) 1928.

Wilson, of New Castle, Pa., removed the parasite through a small incision in the conjunctiva. This filaria was delivered to us and photographed (fig. 2). During March and June of 1941, two worms appeared beneath the right upper palpebral conjunctiva. Neither caused much

Fig. 1.—Calabar swelling on right wrist.

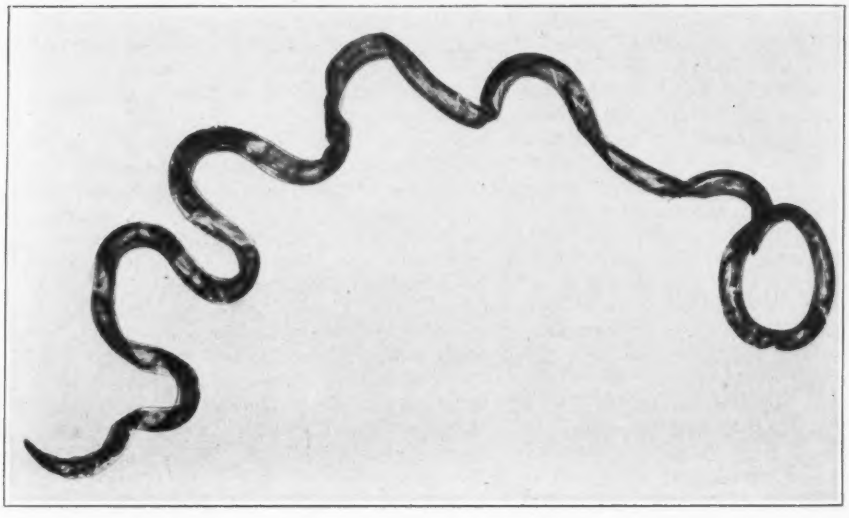

Fig. 2.—Filaria of L. loa, ×6 (actual length 49 mm.). Adult worm removed from the conjunctiva of a missionary returned from the Cameroons, Africa. Note the nuclear structures and the twist at the caudal end.

pain or swelling, a burning or stinging sensation only being noted. Both were removed with the patient under local anesthesia.

The patient first consulted us regarding patches of nondescript dermatitis overlying a persistent nonitching swelling of the ankles. During the period from December 1940 to

February 1941, examinations of the blood were made by the hanging drop method and of a smear stained with Giemsa stain, and a microfilaria (Microfilaria diurna) was demonstrated and photographed (fig. 3). Blood counts at various times demonstrated eosinophilia, the number of eosinophils ranging from 30 to 36 per cent. The patient enjoyed excellent general health during the entire time of her infection.

In an attempt to control the disease mapharsen was given twice weekly during December 1940 and January and February 1941. During this time "Calabar swellings" did not occur, but they reappeared when the drug was given only once weekly. Antimony and potassium tartrate was tried without effect. Various therapeutic measures have been tried by others with questionable results. It is probable that no effective treatment is known. The disease lasts for years, and when infected persons leave endemic areas a spontaneous cure gradually takes place, although adult worms have been removed as long as fifteen years after a patient's departure from the tropics.

Fig. 3.—Microfilaria of L. loa in blood film, × 1,700. Stained by Giemsa's method. Actual length 165 microns. The caudal end is to the right. Note the concentric rings of the larva. Note the clear space and the nuclear bodies, which are anatomically significant. The only successful preparations were made from the blood taken from a Calabar swelling on the forearm in the late afternoon.

COMMENT

From Manson's "Tropical Diseases" [2] we obtained the following information. The parasite is carried by flies, Chrysops dimidiata and Chrysops silacea, both of which are also called the mango fly (fig. 4). In 1891 Manson described the embryonic form of L. loa (Microfilaria diurna), which he recovered from a patient with an adult worm in the eye. He found the microfilaria in the blood and demonstrated that diurna reverses the periodicity of Microfilaria bancrofti, for it is found only during the day, and that the periodicity cannot be reversed by changes in sleeping habits. Development of the larva is slow, and full maturity

2. Manson-Bahr, P. H.: Manson's Tropical Diseases, New York, William Wood & Company, 1931, pp. 569-572.

probably does not occur for several years; thus clinical signs may not appear for several years after infection. The adult worm may be found almost anywhere in the body. It is actively motile and travels about an inch (2.5 cm.) in two minutes. Its presence in the subcutaneous tissues, particularly near articulations, causes the rather sudden appearance of egg-sized or larger swellings accompanied by itching and formication. These swellings, known as "Calabar swellings" may be single or multiple at any one time and usually disappear spontaneously as rapidly as they appear. They never suppurate. Application of heat attracts the worm to the surface, enabling some patients to demonstrate Calabar swellings at will.

The microfilaria (M. diurna) measures 298 by 7.5 microns and is enclosed in a sheath. Its tail is pointed, and it has the same "V" and tail spots as M. bancrofti. The adult male is 30 to 34 mm. long and 0.35 to 0.43 mm. in breadth and tapers toward the tail. The adult female is 20 to 70 mm. long and 0.5 mm. wide. Numerous rounded transulcent protuberances, 12 to 16 microns in diameter and 9 to 11 microns above the surface, mark the body. The alimentary tract extends from the mouth to the middle of the body where the rectum is situated. The worms are ovoviviparous.

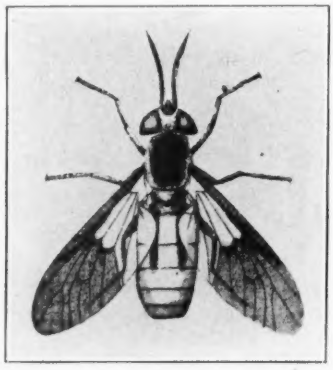

Fig. 4.—The chrysops fly (from Fülleborn, F.: Filiarosen des Menschen, in Kolle, W.; Kraus, R., and Uhlenluth, P.: Handbuch der pathogenen Mikroorganismen, Jena, Gustav Fischer, 1929, vol. 6, pp. 1043-1224).

In 1895 Manson suggested that flies of the genus Chrysops were the carriers, and in 1913 Leiper [3] proved this to be the case. In 1921 [3] the Connals reported on a painstaking study of 2,283 wild flies allowed to feed on 5 native laborers with typical histories and with microfilarias in their blood. The life cycle in the fly is completed in from ten to twelve days. The larvae remain in the stomach of the fly six hours and then migrate to the posterior abdominal muscles in twenty-four hours. Changes in size and shape then occur. The worm sheds its outer coat and takes on a filiform appearance and on the tenth day migrates to the head of the fly and thence to the proboscis. The larvae are deposited on the skin of the host when the fly bites and penetrate the skin in about sixty seconds. Thus man is infected.

Calabar swellings were probably properly credited by Greig [4] to an allergic response to filarial toxins. Ascribed to the same source are urticarial lesions

3. Connal, A., and Connal, S. L. M.: The Development of Loa Loa in Chrysops Silacea and in Chrysops Dimidiata, Tr. Roy. Soc. Trop. Med. & Hyg. **16**:64-89, 1922-1923.

4. Greig, E. D. W.: Notes on Cases of Calabar Swellings with Radiological Observations, J. Trop. Med. & Hyg. **43**:19-21 (Jan. 15) 1940.

with raised red lines lasting from ten to fifteen hours and areas of nondescript dermatitis, such as were seen in our patient. Strong [5] stated that the Calabar swellings are produced by the transitory wanderings of the parasite or the birth of microfilarias, as puncture of a swelling has sometimes shown a microfilaria in the fluid removed. Section of the swelling shows cystic spaces filled with fluid where the tissue has been spread by the movements of the adult parasite. Patients usually enjoy good general health, although some complain of intermittent nausea and headaches. Considerable pain and swelling accompany the passage of a worm beneath the conjunctiva, but no permanent damage is done. Rarely, a more serious complication, such as edema of the glottis, disease of the joints or painful myositis, may occur.

Although we can find no specific statements, we do find inferences in the clinical and experimental literature that the complete cycle of development of the parasite must include the changes noted in the chrysops fly and hence that after departure from endemic areas patients spontaneously recover with removal or death of the adult worms.

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5. Strong, R. P.: Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases, ed. 6., Philadelphia, The Blakiston Company, 1942, vol. 2, pp. 134-139.

HISTOPATHOLOGY OF RAT SKIN IN AVITAMINOSIS A

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Cutaneous lesions due to vitamin A deficiency in man have been described by many authors. The most characteristic feature of such lesions is the formation of keratotic plugs in the hair follicles, with the resulting typically rough, dry skin variously described as phrynoderma, "toad skin" and "nutmeg grater skin."

In experimental animals similar lesions have not been observed. Wolbach and Howe [1] have reported slight but demonstrable atrophy of hair follicles and sebaceous glands but not distended keratinized follicles. Portman [2] has described the loss of subcutaneous fatty tissue and a reduction in the total number of hair follicles present.

The present investigation, however, has revealed that keratotic plugs may readily be induced in rats maintained on a diet deficient in vitamin A and that these plugs are identical with those occurring in the human subject. Access to a large amount of graded material has made possible a detailed study of the processes involved in plug formation.

MATERIAL AND TECHNIC

All animals were obtained at weaning from a colony of Wistar strain albino rats. The litters were weaned onto and maintained on the following diet:

	Per Cent
Coconut cake meal	30
Flaked rice	38
Casein (ashless, extracted)	10
Dried yeast	8
Wheat germ	5
Peanut oil (refined)	5
Salt mixture	4

From the Nutrition Research Laboratory, Lever Brothers & Unilever Limited.

1. Wolbach, S. B., and Howe, P. R.: Tissue Changes Following Deprivation of Fat-Soluble A Vitamin, J. Exper. Med. **42**:753 (Dec.) 1925.

2. Portman, K.: Investigations on the Histological Changes in the Skin of Rats on Various Diets, Acta path. et microbiol. Scandinav. **4**:341, 1927.

The composition of the salt mixture used was as follows:

	Per Cent		Per Cent
Sodium chloride	9.54	Calcium lactate	6.4
Magnesium sulfate	10.1	Ferric citrate	2.4
Sodium phosphate	14.7	Potassium iodide	0.06
Calcium phosphate	28.25	Manganese sulfate	0.05
Potassium phosphate	28.5		

The intakes of vitamin D and vitamin A were under direct control, since each was administered separately to each animal and independently of the diet. The dosage of vitamin D was maintained constant for all rats at 1 international unit per day, while the dosage levels of vitamin A for the several groups were adjusted over a wide range to include, 50, 20, 10, 5, 2, 1 and 0 international units per day. As far as possible, litter mate pairing was adopted, and all rats were weighed at fortnightly intervals.

Small areas of skin were excised from the regions of the abdomen, thorax, shoulder and foot pad of each animal and were fixed in Bouin's fluid. Dehydration of the specimens by diethylene dioxide (dioxane) was followed by embedding in paraffin and sectioning at 5 microns. All slides were stained with either Ehrlich's or Heidenhain's hematoxylin and counterstained with alcoholic solution of eosin.

HISTOLOGIC OBSERVATIONS

The present account is confined to a description of the histologic observations in skin obtained from the region of the abdomen, the structures observed in skin from the various other regions of the body being of an essentially similar nature.

Group 1: Fifty International Units of Vitamin A per Day.—The skin of these rats was found to be in an extremely healthy condition. The cells of the epidermis showed a well graded progression from the cells of the malpighian layer to the thin layer of the stratum corneum, indicating that the processes of growth and keratinization were proceeding normally. The elements of the dermis were well formed, and there were abundant stores of both interfollicular and subdermal fat. The hair follicles were numerous and in close juxtaposition, and the cells at the root of each follicle were in a state of active proliferation. The sebaceous glands were normal. A typical section is illustrated in figure 1 *A*.

Group 2: Twenty International Units of Vitamin A per Day.—The skin from these rats was found to be moderately healthy. There were quite good stores of interfollicular and subdermal fat, but the hair follicles were less numerous and not so well developed as those in the previous group. Occasionally there were suggestions of a slight degree of hyperkeratosis.

Group 3: Ten International Units of Vitamin A per Day.—In this group the fat stores were not so abundant as in the previous groups, although they were still fairly plentiful. Relative hyperkeratosis was

found to occur more frequently. The hair follicles were spaced well apart, and also in some sections there was distention of a few follicles.

Group 4: Five International Units of Vitamin A per Day.—Significant differences between the skins of young and old rats may be observed

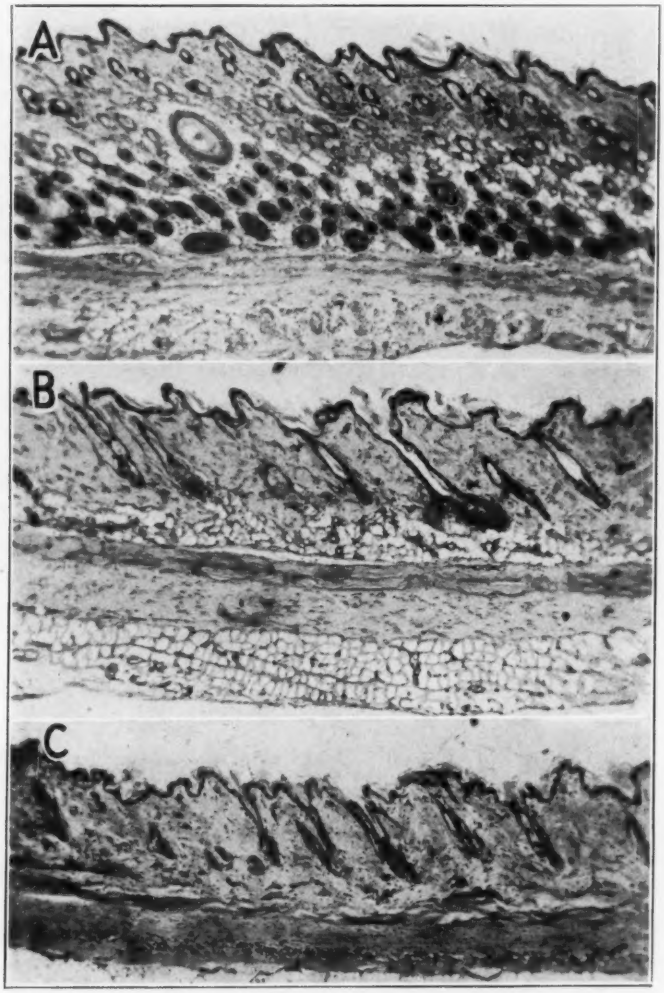

Fig. 1.—Photomicrographs (× 50) of sections of skin from rats given (*A*) 50 units, (*B*) 1 unit and (*C*) 0 units of vitamin A daily for two months. Note the progressive diminution in size and number of hair follicles and the final loss of fat stores.

at this level of vitamin A. One month after weaning the hair follicles were still fairly numerous, although there were a high proportion of distended follicles and some decided hyperkeratosis. At three months after wean-

ing, however, an excessive degree of hyperkeratosis was found at the orifices of some hair follicles. There was also some reduction in the amount of interfollicular fat but no obvious loss in the subdermal depots.

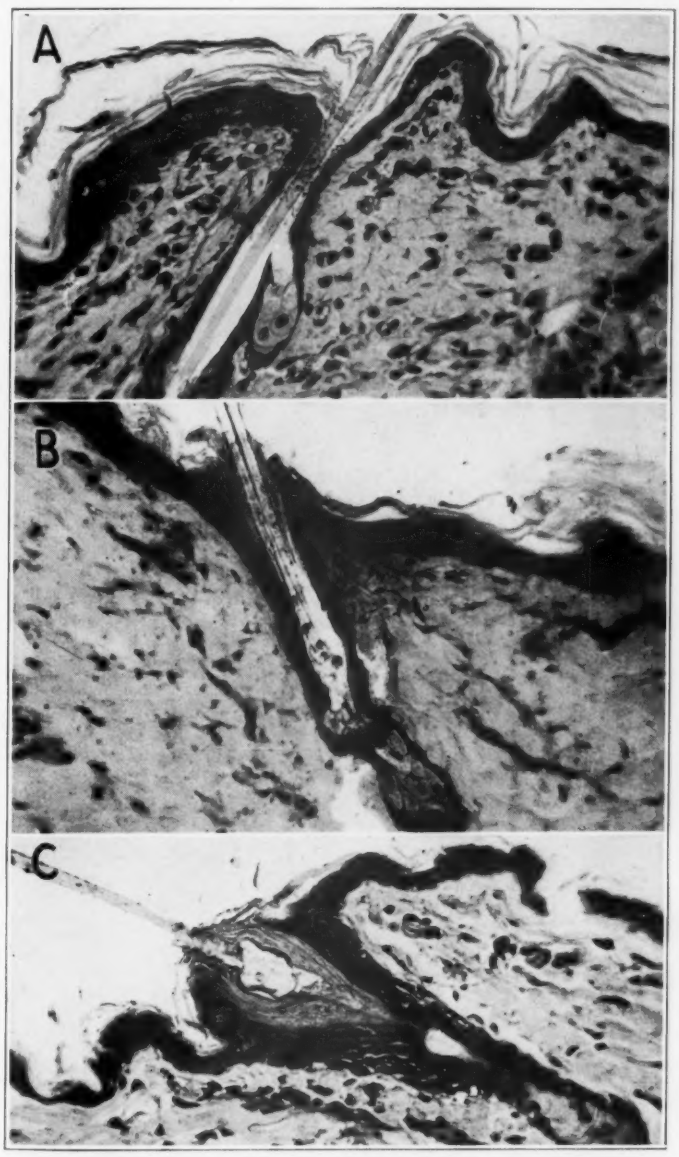

Fig. 2.—Photomicrographs (× 120) of sections of skin from a rat killed on the day of weaning (*A*) and from rats given 2 units of vitamin A daily for one month (*B*) and two months (*C*). Note the progressive keratinization and distention of the follicular orifice.

Group 5: Two International Units of Vitamin A per Day.—In the rats from this group the whole skin was thinner and the abnormalities seen in the previous group were now more acute and were visible at an

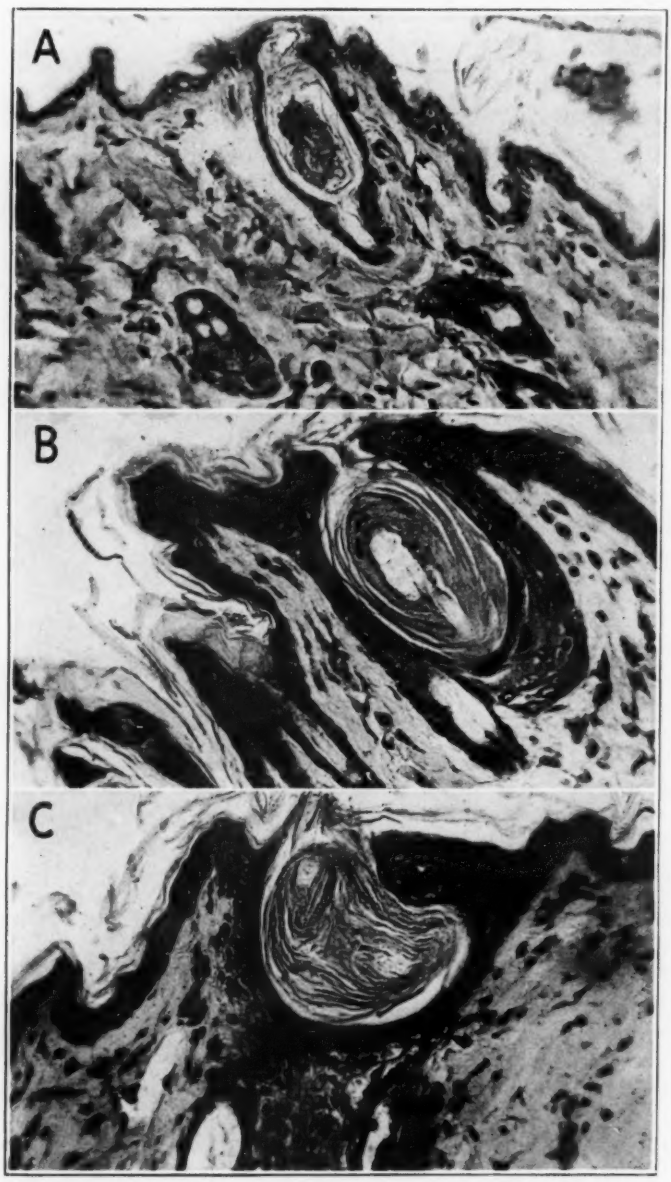

Fig. 3.—Photomicrographs (× 120) of sections of skin from rats (*A*) given 1 unit of vitamin A daily for three months and (*B* and *C*) maintained without vitamin A for one month. Note the continued keratinization and distention of the upper third of each hair follicle.

earlier age. At one month after weaning there was an excessive keratinization at the mouth of many hair follicles (fig. 2 *B*). Two months after weaning in some follicles a relative hyperkeratosis was only just developing, while others were fairly heavily keratinized in their upper thirds. In the latter condition the mouth of the follicle might be considerably distended, while the keratin material protruded beyond the surface of the skin and closely invested the issuing hair (fig. 2 *C*). Nevertheless, the associated sebaceous gland might still be well developed.

Group 6: One International Unit of Vitamin A per Day.—At this level of vitamin A there was considerable keratinization of the upper third of many follicles, associated with a widespread general hyperkeratosis, even at one month after weaning. At two months after weaning both interfollicular and subdermal fat stores were plentiful but the hair follicles were widely spaced (fig. 1 *B*). At three months the fat stores were still rather good, although the upper thirds of many follicles were keratinized and distended (fig. 3 *A*).

Group 7: No Vitamin A Administered.—The youngest rats in this group were those killed actually at the time of weaning, i. e., approximately at the age of 23 days. The remaining animals were killed at intervals of seven, seventeen and twenty-seven days after weaning, while a few were maintained until they appeared to be moribund.

At weaning there was occasionally a slight degree of hyperkeratosis, both follicular and diffuse (fig. 2 *A*), in both bucks and does.

In the case of the bucks, at one week after weaning the degree of follicular hyperkeratosis was advanced to such an extent that the upper thirds of some follicles were already distended by an excessive keratinization. At seventeen days after weaning, a general hyperkeratosis was evident, but the sebaceous glands were healthy and the fat stores moderately good. After one month without vitamin A, the whole skin was somewhat thinner and there was little or no fat. The hair follicles were much less numerous, and many of them were distended. Some follicles had undergone such extensive keratinization that their roots were beginning to atrophy and their upper thirds were completely filled with a stratified mass of keratinous material. Such masses were sufficiently well developed to justify the use of the term "keratotic plug," and a typical plug is illustrated in figure 3 *C*. In such "plugged" follicles, the attached sebaceous gland had always undergone atrophy, in contrast to the normal condition of the sebaceous glands in general.

The sequence of events in the does was similar to that already described. One week after weaning, the keratinization of the upper thirds of some follicles had reached the stage where the mouth of the follicle was again distended with a keratotic plug (fig. 4 *A*). It is important to note, however, that the fat stores might still be plentiful in both

the interfollicular and subdermal regions (fig. 4 *B*), while the sebaceous glands in general had not undergone atrophy but on the contrary appeared to be normal (fig. 4 *A*). At seventeen days and at one month after

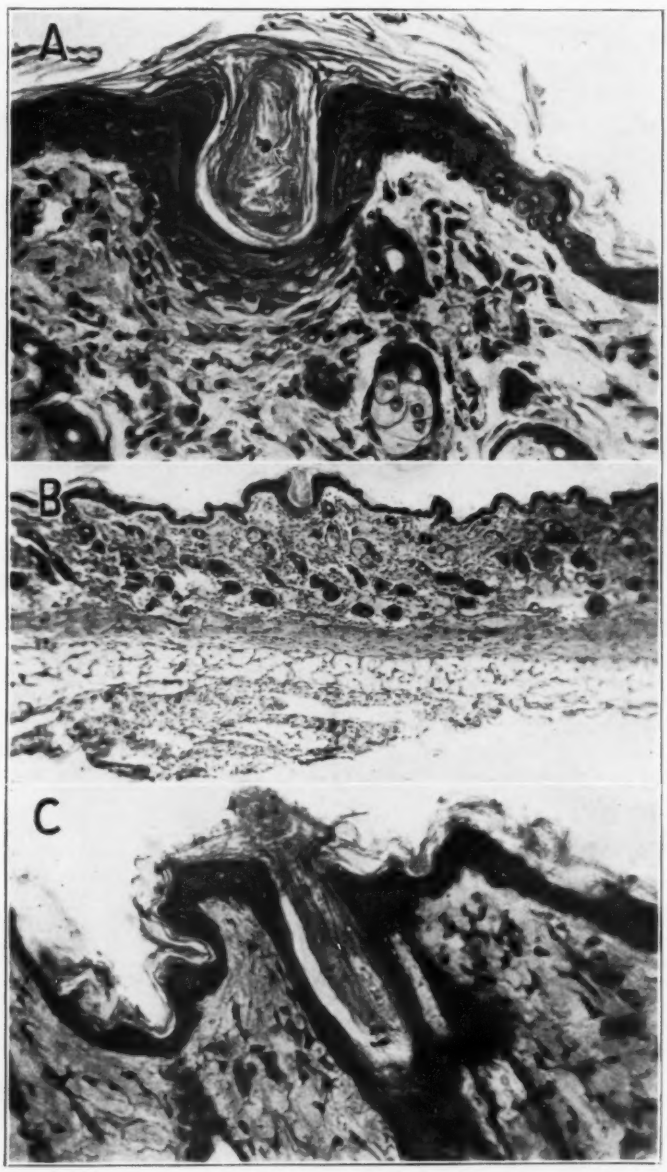

Fig. 4.—Photomicrographs of two sections of skin from rats maintained without vitamin A (*A* [× 120] and *B* [× 501]) for seven days and (*C* [× 120]) for one month.

weaning, the fat stores were beginning to be depleted and keratotic plugs were again found (fig. 3 *B*). In 1 specimen there was an interesting modification of one of the plugs which projected appreciably beyond the surface of the skin in the form of a horny spine (fig. 4 *C*).

Although under the conditions of the experiment bucks appeared to become moribund after about six weeks and does after ten weeks, the appearance of the skin was fundamentally the same in both. The whole skin was thin and atrophic; there was no interfollicular fat and hardly any subdermal fat, and the sebaceous glands had atrophied (fig. 1 *C*).

COMMENT

In the series of experiments herein recorded, there are two variable factors: the dose of vitamin A and the duration of the treatment (i. e., the increasing age of the animal). These factors, however, do not operate independently but are interrelated.

At any given age, progressively decreasing doses produce progressively more decided lesions. For example, the sections shown in figure 1 were all prepared from rats which had been under experimental treatment for about two months after weaning, the doses of vitamin A being respectively 50, 1 and 0 international units per day. It will be seen that there is a progressive diminution in the thickness of the skin and in the size and number of the hair follicles. Finally, the fat stores are completely lost and the sebaceous glands atrophy.

Similarly, at a given suboptimal dosage level, increasing age results in increased incidence and severity of lesions. For example, only a slight degree of hyperkeratosis exists in the youngest rats, which were killed on the day of weaning (fig. 2 *A*). After one month on a dosage level of 2 international units of vitamin A per day, the upper thirds of some follicles may be keratinized but are not appreciably distended (fig. 2 *B*). After two months' treatment, the degree of keratinization of the upper thirds may be sufficient to have caused a decided distention of the follicular orifice (fig. 2 *C*).

It is important at this juncture that some distinction should be drawn between the primary lesions, for which the absence of vitamin A is specifically responsible, and the secondary sequelae, which are not specific. The excessive keratinization of epithelial tissues is undoubtedly a direct result of vitamin A deficiency. A state of partial deficiency also effects a considerable reduction in the total number of hair follicles, but the actual cessation of growth of hair is probably due to purely mechanical obstruction caused by the incipient plugging at the mouth of each individual hair follicle.

Similarly, interference with the outflow of sebum may itself induce atrophy of the sebaceous gland concerned. This point is emphasized

because some authors, for example Steffens and Sheard,[3] have stated that deficiency of vitamin A causes atrophy of the sebaceous glands. In the present investigation it is shown that a state of deficiency sufficient to have caused such extreme lesions as keratotic plugs is still not sufficient to have induced the atrophy of sebaceous glands not attached to the atrophied follicle concerned (fig. 4 *A*). In fact, general atrophy of the sebaceous glands is usually contemporaneous with complete loss of the interfollicular and subdermal stores of fat, a condition found only in the atrophic skin of rats which have been permitted to become moribund (fig. 1 *C*). Consequently it may be concluded that total atrophy of the sebaceous glands is not specifically characteristic of vitamin A deficiency. Portman[2] has suggested that atrophy of sebaceous glands and loss of fat stores are the result of protracted inanition. According to Smith, Smith and Callaway,[4] maintenance of the sebaceous glands in the rat is dependent on an adequate supply of vitamin G in the diet, and administration of vitamin A is without effect.

A search of the literature has failed to reveal any previous report of the appearance of keratotic plugs induced by vitamin A deficiency in experimental animals, and hence an itemization of the stages involved in the process of plug formation seems justified. Initially there is a slight general or diffuse hyperkeratosis (fig. 2 *A*), which is followed by an increased keratinization of the epithelium forming the upper third of the hair follicle (fig. 2 *B*). Subsequently this follicular hyperkeratosis becomes so intense that the orifice of the follicle may become partly distended with a bulbous mass of keratin (fig. 2 *C*), although the hair itself continues to grow and the sebaceous gland may be healthy. Further increase in the degree of the keratinization (fig. 3 *B*) causes a decided distention of the follicular orifice and impedes both the growth of the hair and the outflow of sebum. Eventually a distinct plug, consisting of a stratified mass of keratin, is formed (figs. 3 *C*, 4 *A* and 4 *B*); the hair follicle becomes completely disorganized, and the attached sebaceous gland atrophies.

Keratotic plugs occurring in human skin have frequently been reported; a complete review was presented in a recent paper by Frazier and Hu.[5] These authors[6] were also the first to suggest that the lesion

3. Steffens, L. F., and Sheard, C.: Dark Adaptation and Dietary Deficiency in Vitamin A, Am. J. Ophth. **23**:1325 (Dec.) 1940.

4. Smith, S. G.; Smith, D. I., and Callaway, J. L.: Dysfunction of the Sebaceous Glands Associated with Peflagra, J. Invest. Dermat. **4**:23 (Feb.) 1941.

5. Frazier, C. N., and Hu, C. K.: Nature and Distribution According to Age of Cutaneous Manifestations of Vitamin A Deficiency: A Study of Two Hundred and Seven Cases, Arch. Dermat. & Syph. **33**:825 (May)) 1936.

6. Frazier, C. N., and Hu, C. K.: Cutaneous Lesions Associated with a Deficiency of Vitamin A in Man, Arch. Int. Med. **48**:507 (Sept.) 1931.

was characteristic of vitamin A deficiency, and subsequently Radhakrishna Rao [7] recorded similar conclusions. Careful analysis reveals a remarkable degree of similarity between the plugs described by these authors and those now observed in the rat and illustrated in figures 3 *C* and 4 *A*. Again, the type of plug shown in figure 4 *C* is exactly comparable with the protruding horny spine described by Pemberton.[8]

From consideration of their histologic and etiologic identity, there can be no doubt that the keratotic plugs induced by vitamin A deficiency in the rat and in man are completely homologous.

SUMMARY AND CONCLUSIONS

1. Under the conditions of the experiment, rats receiving 50 international units of vitamin A and 1 international unit of vitamin D per day possess normal healthy skins free from any detectable lesions.

2. In avitaminosis A, the skin of rats exhibits certain characteristic lesions, viz., follicular hyperkeratosis leading to the formation of keratotic plugs in the hair follicles.

3. From consideration of their histologic and etiologic identity such keratotic plugs induced in the rat are completely homologous with those previously reported in man.

4. Cessation of hair growth and atrophy of individual sebaceous glands are caused primarily by the mechanical obstruction of the hair follicles consequent on the formation of keratotic plugs.

5. General atrophy of sebaceous glands and loss of dermal fat stores are not specific sequelae of vitamin A deficiency.

The directors of Lever Brothers and Unilever Limited gave permission to publish these studies. Dr. H. Wilkinson gave suggestions and advice during the course of the experimental work.

7. Radhakrishna Rao, M. V.: Studies on Vitamin A Deficiency: Histopathology of the Skin in Human Keratomalacia, Indian J. M. Research **25**:39 (July) 1937.

8. Pemberton, J.: Follicular Hyperkeratosis: A Sign of Malnutrition? Lancet **1**:871 (May 11) 1940.

ICHTHYOSIFORM ATROPHY OF THE SKIN IN HODGKIN'S DISEASE

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Atrophy, keratoses and pigmentation are among the nonspecific cutaneous manifestations described in Hodgkin's disease.[1] Nanta and Chatellier [1g] spoke of ichthyosiform hyperkeratoses as exceptional cutaneous reactions of the general intoxication.

REPORT OF A CASE

D. G., an athletically built 24 year old American of Jewish extraction, a salesman and a football player, was seen in July 1938 at the tumor clinic of the Rhode Island Hospital because of enlarged supraclavicular lymph nodes.

His past history revealed that on May 4, 1936 he was seen at the tumor institute of the Swedish Hospital, Seattle, because of a tumor mass involving the entire right supraclavicular region and right lower region of the neck. A biopsy was made at the laboratory of the Marine Hospital and of the Swedish Hospital, and the diagnosis was Hodgkin's disease. He received roentgen therapy, and on May 19 the mass was reported as having entirely disappeared and the wound made when material was obtained for biopsy was well healed. On October 9 he was reported in excellent general condition with no evidence of disease.

At the tumor clinic of the Rhode Island Hospital, on July 8, 1938, besides the enlarged supraclavicular lymph nodes, a roentgenogram showed a mass in the mediastinum consistent with lymphoma. Because of the large size of the mass further roentgen treatment was advised.

On September 8 a lymph node was excised from the neck, and the diagnosis of Hodgkin's disease was confirmed (Dr. B. E. Clarke). On Feb. 15, 1939 a roentgenogram showed no remnants of the mediastinal tumor and the patient had no complaints. In many subsequent visits to the tumor clinic of the Rhode Island Hospital he was found well. In May 1940 a node appeared in his pelvic region and roentgen rays were administered to that region. In October 1940 the swelling

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From the Department of Dermatology and the Tumor Clinic of the Rhode Island Hospital.

1. (*a*) Cole, H. N.: The Cutaneous Manifestations of Hodgkin's Disease: Lymphogranulomatosis, J. A. M. A. **69**:341 (Aug. 4) 1917. (*b*) Fox, H.: Lymphogranulomatosis of the Skin in Hodgkin's Disease, Arch. Dermat. & Syph. **2**:578 (Oct.) 1920. (*c*) Fraser, J. F.: Cutaneous Manifestations in Hodgkin's Disease, ibid. **46**:587 (Oct.); 769 (Nov.) 1942. (*d*) Mariani, G.: Stato attuale delle nostre conoscenze sulle leucemidi cutanee, Gior. ital. d. mal. ven. **61**:63, 1920. (*e*) Martinotti, L.: Le manifestazioni cutanee nelle emoblastosi, ibid. **61**:91, 1920. (*f*) Miller, H. E.: Lymphogranulomatosis Cutis-Hodgkin's Disease, Arch. Dermat. & Syph. **17**:156 (Feb.) 1928. (*g*) Nanta, A., and Chatellier, L.: Lymphogranulomatoses chéloidiennes pseudo-leucoplasiques: le chancre lymphogranulomateux, Ann. de dermat. et syph. **6**:682, 1925.

reappeared on the right supraclavicular region and more roentgen rays were administered. In June 1941 the patient began to notice loss of appetite and loss of weight.

In November 1941 the patient appeared to have lost more weight. He complained of not feeling too strong, but otherwise he was well and still did not believe in the prognosis of his case. At this time a peculiar change was noticed on his skin. Dr. B. E. Clarke, who kept him under close observation, and Dr. Philip

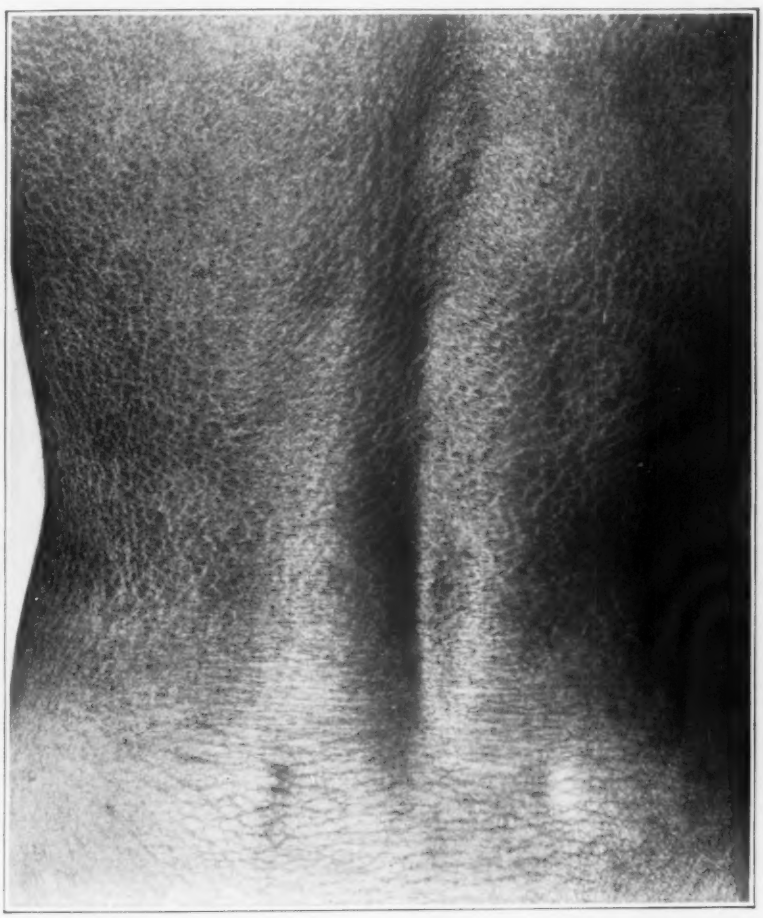

Fig. 1.—The back of the patient, showing the diffuse ichthyosiform atrophy of the skin.

Batchelder, who administered the therapy for over three years, noticed the change from an apparently normal skin to a dry, wrinkly, hyperpigmented one. The change, according to the patient, took place in the previous six weeks. The cutaneous surface was made of rhomboidal scales with flaky edges, such as are seen in mild ichthyosis or in senile ichthyosis. The palms and soles were also dry, with palmar lines as seen in dry chronic eczema of the palms.

He emphatically stated that his palms were previously always of a normal appearance. No erythema or edema was noticeable. The patient did not com-

plain of itching or discomfort. He was just back from Florida, where he had "baked" in the sun and paid no attention to the discoloration and texture of his skin.

Material for biopsy was taken from the right scapular region. It took several weeks for the wound to heal. Dr. B. E. Clarke's report was as follows: "There is a thick horny layer on the surface of the epidermis. Other layers of the epidermis are thin. The papillae are considerably ironed out. In places there is considerable brown pigment in the basal layer and elsewhere none. The corium is quite dense and hyaline. There are in it two or three hair follicles and a few coil glands.

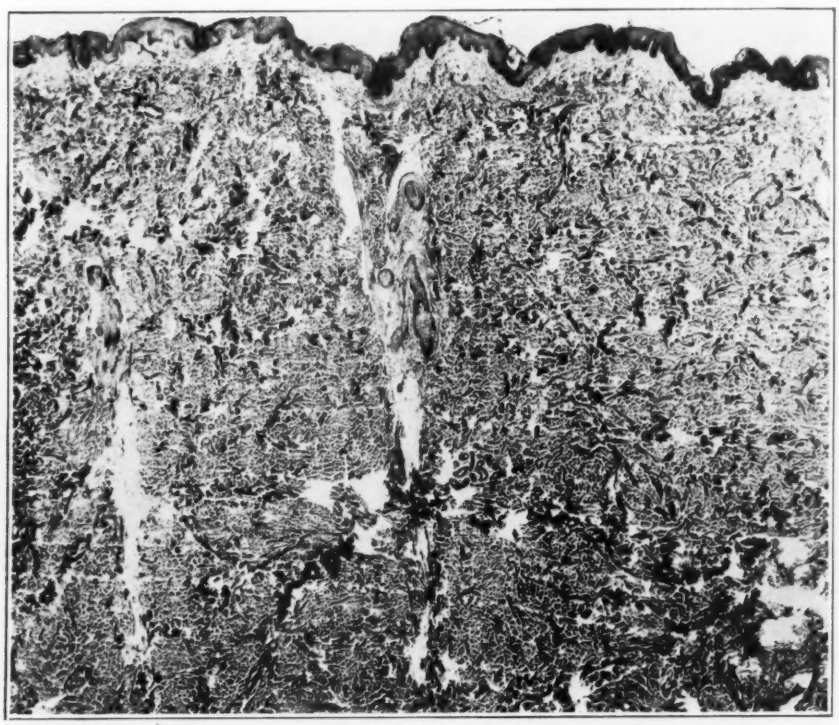

Fig. 2.—A low power photomicrograph, showing mild hyperkeratosis, thinning and flattening of the epidermis, absence of sebaceous glands and no evidence of lymphogranulomatosis.

No sebaceous glands are seen in three sections. Diagnosis: atrophy and hyperkeratosis of the skin."

Dr. H. Montgomery, Rochester, Minn., examined the section and wrote as follows: "The absence to partial formation of the stratum granulosum supports a diagnosis of ichthyosis, as does the increased melanin pigmentation. There is no evidence of Hodgkin's disease."

The patient began to fail rapidly. On Sept. 19, 1942 he was admitted to the Rhode Island Hospital because of weakness, extreme emaciation and urinary incontinence. His skin was dry and flaky. His face was emaciated and fixed, as in scleroderma.

He died on October 22. An autopsy was not permitted.

SUMMARY AND CONCLUSIONS

A case has been reported in which ichthyosiform atrophy of the skin developed in a patient with Hodgkin's disease eleven months before death, while he was apparently in good health.

This complication is one of the many cutaneous nonspecific toxic manifestations of lymphogranulomatosis, perhaps heralding the final stage of the disease.

122 Waterman Street.

CHRONIC SCARRING PSEUDOFOLLICULITIS OF THE NEGRO BEARD

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A peculiar condition involving the bearded part of the face and neck is present in a considerable percentage of Negro men but has attracted little attention in the medical literature.

CLINICAL PICTURE

The fully developed picture is seen in the middle-aged or older men. Usually the skin of the lower part of the cheeks is affected most, but the changes may extend to the chin, the sides of the neck and the upper regions of the cheeks, in the order named.

There are numerous superficial crisscross grooves, each from 1 to 3 mm. long and a little wider than a hair's breadth. Many of these grooves contain a hair which lies in them parallel to the surface of the skin. Other grooves are empty and have the appearance of superficial scars. In between, and usually more frequent along the rim of the mandible and on the sides of the neck, there are firm skin-colored obtuse papules from pinhead sized to hempseed sized, each of which is pierced obliquely by a hair. These hairs extend in a flat curve almost parallel with the surface for a short distance and then disappear back into the skin. They thus form a loop similar to a shoe lace which is led out of one eyelet and down into another.

On lifting the loop with a forceps one finds that the papule does not contain the root, but the free end of the hair, which sometimes has buried itself in the skin for as much as 3 mm. These hairs always have sharp shaved-off ends (fig. 9*c*). Younger men have fewer "grooves" and more papules. In some cases, inflammatory changes are pronounced, many of the papules have pustular centers and the skin of the entire region affected may be slightly swollen and have a darker livid hue, due to the addition of inflammatory redness to the normal pigmentation.

The condition is common. It is well developed in at least 10 per cent of the Negroes, and minor degrees of it may be found in many more. The clinical picture is characteristic. It is evidently due to hairs growing back into the skin and causing irritation, inflammation

From the Department of Dermatology, Monroe Hospital.

and finally scarring. Most of the men accept the condition as something inevitable, and only a few seek medical advice. On being questioned, however, many will say that their beard has been causing them much discomfort for a great number of years. As the men grow older, the hairs appear to undergo atrophy, and finally nothing is left but scars. Thus the condition, while extremely chronic, is self limited.

I became interested in this variety of "ingrown hairs" and have considered the subject worthy of publication for three reasons: First, there is a characteristic clinical picture; second, there is a definite pathogenic mechanism based on the peculiar anatomy of Negro hair, and, third, treatment of the patients who apply for medical care is a real problem. It will suffice to cite 1 case:

R. W., a Negro 25 years of age, complained of trouble with his beard since he started shaving, at the age of 12 years. He thought it was getting a little better, but the ingrown hairs caused much burning and itching, particularly in the summer, and disturbed him in his work. He shaved about once a week and dug out the ingrown hairs. Some of these appeared to be dead, and pulling them out did not cause pain.

Examination showed a strongly built man of medium brown color with typical Negro features and kinky black hair. The beard was well developed, and there was a moderate amount of curly hair on the chest. The region from 1 inch (2.5 cm.) above the margin of the mandible down the sides of the neck as far as the beard extended had a dark purplish hue, and there were numerous hempseed-sized papules, some of which had a central pustule (fig. 1). Each one of these papules was pierced by a hair, which could be lifted out and showed the beveled point of the shaved hair while the other end was buried in a hair follicle 1 or 2 mm. away. The follicles showed no signs of inflammation; the hairs were firmly fixed, and their roots appeared normal when they were pulled out. Because of some general swelling of the skin, these hair loops often lay in shallow grooves, but they were never covered by the epidermis or horny layer. The papules were most numerous beneath the mandible; they became sparse higher up on the cheeks. Here, there were many narrow depressed scars, 1 to 3 mm. long, extending in all directions. This is visible in figure 1 *B* and is demonstrated particularly well in a dermatogram (fig. 2). Some of the grooves contained hairs; in others no trace of hair could be found.

A specimen for biopsy was taken from the left side of the neck. The patient then was given an epilating dose of filtered roentgen rays to each of two fields, taking in the affected areas of the sides of the neck and the lower part of the cheeks. Effluvium capillorum followed seventeen days later. The patient felt greatly relieved and was advised to shave daily as soon as the hairs would grow back.

PATHOLOGY AND PATHOGENESIS

Among white patients the term "ingrown hairs" is used ordinarily for several types of abnormal growth, which have in common that the hair does not erupt properly from the follicular opening:

1. The most common and entirely harmless type is found in cases of mild ichthyosis and hyperkeratosis suprafollicularis on the trunk and

extremities. Here, the lanugo hairs do not have the power of piercing the thickened horny layer and curl up beneath it in a spiral. One can free the hair easily by scraping away the hyperkeratosis.

Fig. 1.—Patient with chronic scarring pseudofolliculitis.

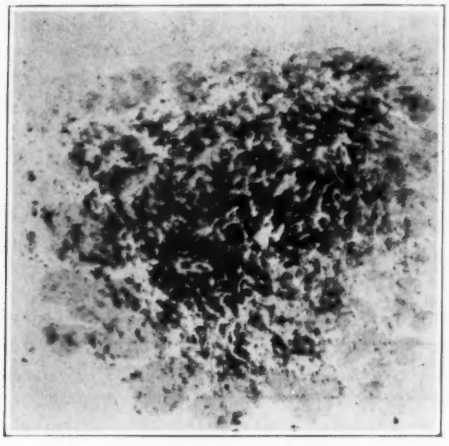

Fig. 2.—Ink print (*dermatotypie*, M. Oppenheim) of the left cheek of patient described in text. Crisscross pattern of white grooves is due to loop hairs and scars. A few ring-shaped imprints are due to inflammatory papules.

2. Much more annoying are those ingrown hairs of the beard, found in cases of severe acne or sycosis, in which inflammation and scarring have altered the direction of the hair roots. The hairs then

curl up in the skin and produce foreign body inflammation. They may reach considerable length and often require surgical removal.

3. A third variety is well known to many men in whom there is a tendency for small inflammatory papules and pustules to develop on

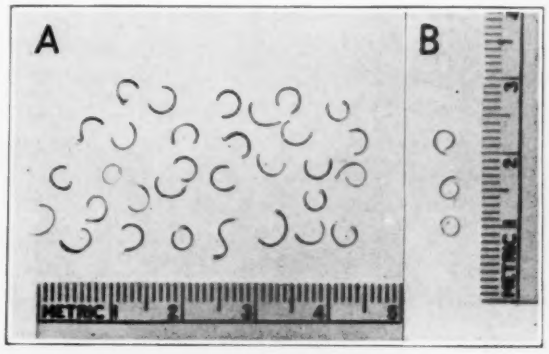

Fig. 3.—Hairs collected from case 1 after roentgen ray epilation. *A*, normal hairs. *B*, three loop hairs, which are thinner and smaller than most of the hairs in *A*.

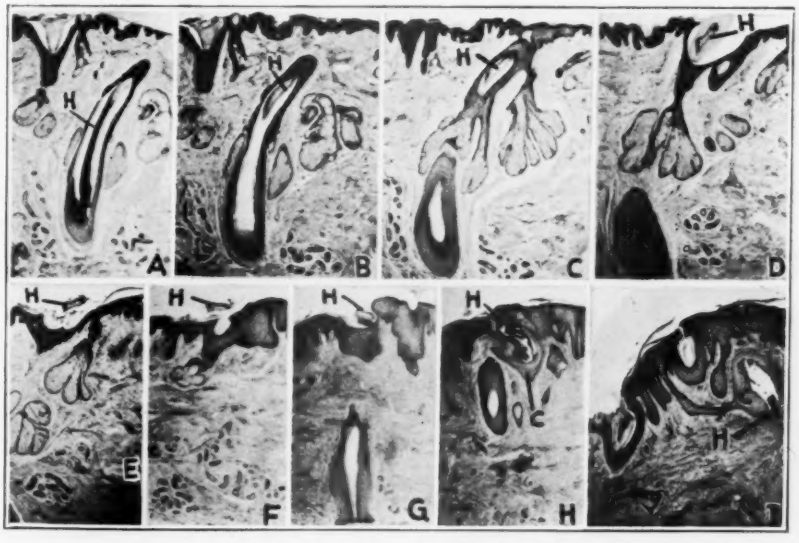

Fig. 4 (case 1).—Nine sections selected from a total series of 212 (hematoxylin-eosin; 20 microns). The upper row shows sections of the semicircular hair follicle, which is cut once in *A* and *B* and twice in *C* and *D*. *H* indicates the hair, which emerges from the skin in *D*. *E* and *F* show cross sections of the hair loop above the horny layer. *G* shows the hair entering the skin. *H* shows it within the epithelial cuff (*c*). *I* shows it leaving the cuff and extending bare into the corium. Note the epidermal hypertrophy and inflammatory infiltration in *F* to *I*.

the sides of the neck after shaving. The lesions contain short living hairs which are visible directly under the epidermis without erupting. This is the only type which bears a certain clinical resemblance to the disease being discussed.

The "loop hairs" of the Negro beard differ from all these types because they erupt normally but grow back into the skin.

In order to gain complete insight into the pathology of the disturbance, serial sections were cut of the biopsy specimen (fig. 4) from the case reported (case 1) and of three small biopsy specimens from a similar case (case 2). In addition, small pieces of skin containing "ingrown hairs" of the third variety mentioned were removed from the necks of 2 white men. One of these (case 3) had fair straight hair and the other (case 4) black curly hair. These biopsy specimens also were cut into serial sections (fig. 5). Dr. Felix Pinkus then made large scale plastic reconstructions of four hairs, one from each case (fig. 6).

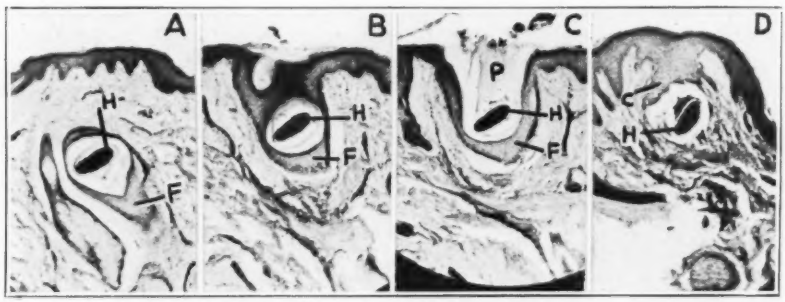

Fig. 5 (case 4).—Four sections selected from a total series of 112 (hematoxylin-eosin; 20 microns). *A* shows a cross section of the slanting hair follicle (*F*) at the level of the sebaceous gland. *B*, junction of the follicle with the epidermis. *C*, the follicular orifice is filled with a horny plug (*P*), while the hair (*H*) remains in the depth. *D*, the hair lies bare in the cutis surrounded by inflammatory reaction. *c* denotes part of the epithelial cuff which accompanies the ingrown hair.

These studies made clear the fundamental difference of the mechanism of "growing in" of the straight or curly hairs of white men and the kinky hairs of Negroes.

The root of the white man's hair is straight or slightly curved and extends down into the skin at a more or less acute angle. At some parts of the body, notably on the neck and scrotum, the roots are embedded at such a bias that their slanting course may be seen through the superficial layers of the skin with the naked eye. Plastic reconstruction (figs. 6 A and B and 7) makes it evident that an ingrown hair is a shaved-off hair which pierces the side wall of the follicle and enters the cutis instead of emerging at the follicular opening. It

seems that this happens when a slanting hair has been shaved off so close that the attending pull on the skin causes its free end to slip below the surface. As may be seen in figure 7, the follicle curves upward just below the epidermis. The bristly hair, which has lost its natural soft tip, does not follow this curve but is pushed through the side of the follicle by the pressure of growth from the root.

The root of the negroid hair, on the other hand, forms a more or less complete semicircle in the skin. The follicle of a hair of the beard

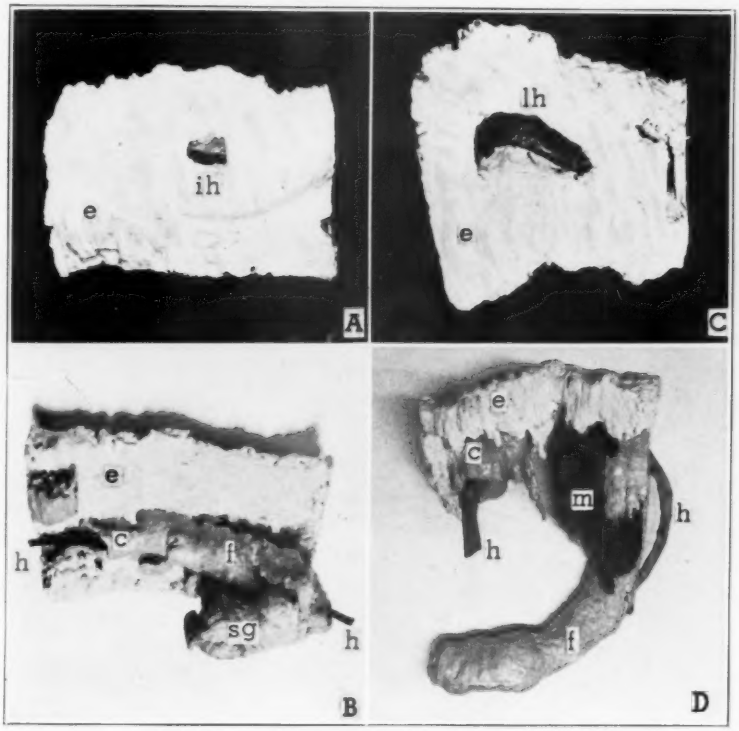

Fig. 6.—Photographs of two three dimensional models of ingrown hairs by Dr. Felix Pinkus. *A* and *B*, white person (case 4). *C* and *D*, Negro (case 1). *A* shows the surface of the epidermis (*e*) without the horny layer; *ih*, ingrown hair in the depth of the follicular orifice. *B* shows the same model from below; *f*, follicle, the deepest part of which has been removed in order to show the hair (*h*) at the right; *sg*, sebaceous gland; *c*, epithelial cuff around the ingrown part of the hair at the left. *C* shows the surface of the epidermis (*e*) and hair (*lh*) forming a loop above the surface. *D* is a side view of the same model, showing the semicircular follicle (*f*), part of which has been removed in order to show the hair (*h*) at the right; *m*, arrector muscle; *c*, epithelial cuff sheathing the ingrown part of the hair which extends into the corium at the left and almost completes the circle.

in particular is often curved in such a manner that the hair leaves the skin in a slanting direction and, following its natural curve, grows back against the surface of the skin. Hairs which have their natural soft tip are deflected and curl up in a horizontal spiral. If the sharp end of a shaved hair, however, is permitted to grow long enough it pushes its way into the skin, like an ostrich burying its head. Once inside, it

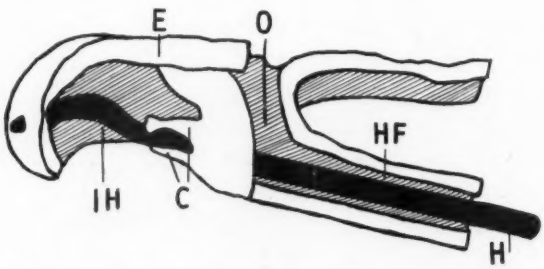

Fig. 7.—Schematic drawing based on large scale reconstruction of white men's ingrown hairs by Dr. Felix Pinkus. The horny layer and connective tissue are omitted. *E* indicates epidermis; *HF*, hair follicle; *O*, follicular orifice; *H*, hair; *IH*, ingrown part of the hair, and *C*, epithelial cuff sheating the ingrown hair which has pierced the side wall of the follicle.

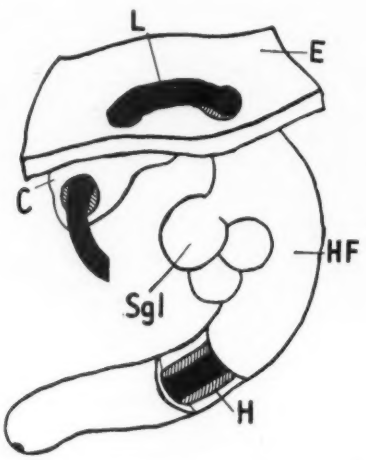

Fig. 8.—Schematic drawing based on large scale reconstruction of negroid loop hairs by Dr. Felix Pinkus. The horny layer, connective tissue and hair muscle are omitted. *E* indicates epidermis; *HF*, hair follicle; *Sgl*, sebaceous gland; *H*, hair in the follicle; *L*, hair loop, and *C*, epithelial cuff sheathing the hair after it has buried itself in the skin.

grows deeper, completing the spiral in a vertical direction (figs. 6 *D* and 8). One question remains unsolved; that is whether the loop hair enters the opening of another follicle. Hairs often disappear in the

skin so smoothly at either end that one cannot be sure which end is the root until one lifts the hair. Histologic examination and plastic reconstruction did not answer this question completely. The free end of the hair is surrounded to a fairly deep level by an epithelial cuff which even may show root-sheath-like cornification (fig. 9 *A* and *B*). The tip, however, extended free into the corium in all cases examined and was surrounded by foreign body inflammation. No remnants of an oil gland, muscle or hair bulb were found on this epithelial pouch. It may well be that the sheath is furnished by the epidermis, which tries to epithelize the defect. A similar cuff was found forming around

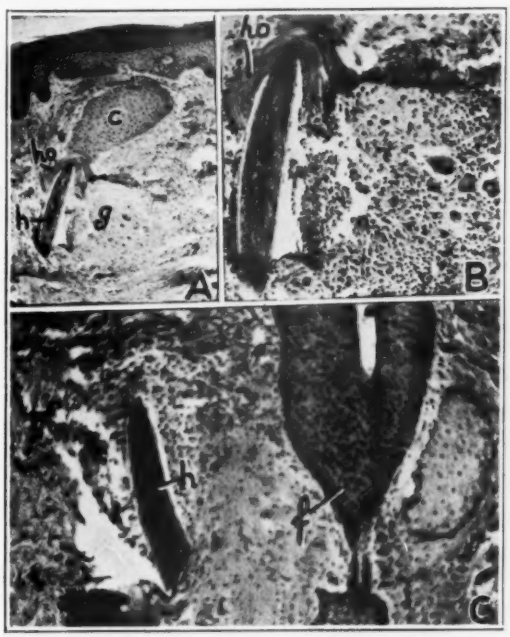

Fig. 9 (case 2).—*A* shows part of the ingrown hair (*h*) in the cutis surrounded by foreign body granulation tissue (*g*) with giant cells. Above it is part of the epithelial cuff (*c*), which shows root-sheath-like horny layers (*ho*). *B*, detail of *A*. *C*, the end of the hair (*h*), which has been cut obliquely by the razor, has pierced the corium to a depth where it is at the level of the sebaceous gland of a neighboring follicle (*f*).

the ingrown hairs of white men, where it is derived from the follicular epithelium or from the epidermis (figs. 6 *B* and 7).

The ingrown hairs of the third variety and the loop hairs of Negroes thus have a common pathogenic factor. In each instance shaving is a prerequisite. However, in the case of straight hairs, shaving too close causes trouble while shaving not close enough permits kinky hairs to grow in.

Three loop hairs and a large number of normal ones were removed from the cheeks and neck of the patient reported on (case 1) at the time of the effluvium capillorum due to roentgen rays. Figure 3 shows that the loop hairs were thinner and weaker than most of the normal ones. This observation supports the impression that these hairs gradually die out. Pressure atrophy is the most likely explanation, since no inflammatory changes are present around the root.

REVIEW OF THE LITERATURE

A search of the literature revealed a strange absence of references to this condition. The excellent papers of Morison,[1] Fox[2] and Hazen[3] contain no indication that the peculiar picture caught the attention of these keen observers. Neither does a recent series of articles by Loewenthal[4] mention this condition.

The only remark that might be construed as referring to this subject is one by Wise[5] in the discussion of Hazen's second paper. Wise stated that "mild and often severe sycosis of the bearded portion of the neck is common, much more common than it is in white persons." Hazen agreed with this observation.

Dubreuilh[6] is the only author who made 2 cases of the early acute stage of the disease the subject of a discussion in which he brought out most of the important points of clinical appearance and pathogenesis. In 1922 he published a paper entitled: "Eruption papuleuse de la barbe causée par le recourbement des poils." One of his patients was a French mulatto soldier from Guadeloupe; the other patient's race was not mentioned, and presumably he was a white person. The second one offered some atypical findings; many of the hairs did not emerge from the skin at all but could be seen under the horny layer. One must also consider that persons with negroid hair are found among the white Mediterranean population. Dubreuilh distinguished the condition in his cases clearly from ingrown hairs which result from difficulties of the hair to erupt. He recognized that the follicle is normal and that the papular or pustular inflammation is at the point where the normally

1. Morison, R. B.: Personal Observations on Skin Diseases in the Negro, M. News **53**:439-444, 1888.

2. Fox, H.: Observations on Skin Diseases in the Negro, J. Cutan. Dis. **26**:67-79, 1908.

3. Hazen, H. H.: (*a*) Personal Observations upon Skin Diseases in the American Negro, J. Cutan. Dis. **32**:705-712, 1914; (*b*) Syphilis and Skin Diseases in the American Negro: Personal Observations, Arch. Dermat. & Syph. **31**:316-323 (March) 1935.

4. Loewenthal, L. J. A.: Diseases of the Skin in Negroes, J. Trop. Med. **39**:209, 250 and 260, 1936; **40**:266 and 277, 1937; **41**:187, 1938.

5. Wise, F., in discussion on Hazen.[3b]

6. Dubreuilh, M. W.: Bull. Soc. franç. de dermat. et syph. **29**:80-81, 1922.

erupted hair grows back into the skin. He stressed the point that the free end of the hair always shows evidence of having been cut by the razor. In his second case the trouble started after the young man had discontinued shaving for several weeks because of illness.

DIAGNOSIS

Thus this condition, though of no great practical importance, is a well defined pathogenic entity running a definite course and leading to a typical clinical picture which is easily diagnosed and differentiated from other involvement of the beard. Because the individual lesions are always pierced by a hair and on first sight seem to be of follicular origin and because scar formation is a characteristic and inevitable part of the picture, the name chronic scarring pseudofolliculitis appears appropriate.

There are few cutaneous diseases from which this one must be differentiated. Sycosis vulgaris is the most important one. Most observers agree that sycosis is fairly uncommon in Negroes, a point which is particularly interesting in view of the wide distribution of pseudofolliculitis, which would appear to give an ideal background for staphylococcic infection. The follicular pustules of sycosis are easily differentiated from pseudofolliculitis by the test of lifting the hair out of the lesion. It might be mentioned in this connection that I have never seen keloidal changes resulting from pseudofolliculitis. This is noteworthy, since folliculitis keloidalis of the neck is particularly common among Negroes on the basis of chronic follicular infection and in view of the inclination of the Negro skin to form keloids.

Acne vulgaris should not offer any difficulties of differentiation. It is not common among Negroes.

One rare condition which might be confused with the late stages of pseudofolliculitis cicatrisans is atrophoderma vermiculatum. Its localization is typically the upper part of the cheeks, where the present condition is found only when it is extensive, in which case the widespread distribution of lesions and the characteristic features of the hairs should make a diagnosis easy.

THERAPY

It is an interesting thought that a normal racial characteristic under the influence of a beautifying procedure which is one of the oldest inventions of mankind and is practiced from the lowest to the highest level of civilization is responsible for a "disease" which causes considerable discomfort and permanent disfigurement to a large portion of this racial group.

Still, the most rational approach to cure and prevention of pseudofolliculitis appears to be the advice of shaving daily and as closely as possible. If a man prevents his beard from growing so long that hairs

can curve back into the skin, irritation and its sequels will be avoided. This advice, however, is not easily carried out, because the Negroes' hairs curve in various directions. This and the fact that many hairs are embedded in grooves make it hard to catch all of them with the razor.

The hairs which have grown in must be epilated of course. When the condition is severe, with many loop hairs and much inflammation, temporary epilation by roentgen rays is indicated. It relieves the distressing symptoms of itching, stinging and burning within a few days, and it gives the patient a chance to let his skin recover from the injury.

Dubreuilh found moist packs and sulfur ointments beneficial in his cases, sulfur probably acting as an antiseptic as well as a keratolytic agent. One might consider the use of depilatory creams.

SUMMARY

Attention has been called to a peculiar disease which affects the bearded skin of a large percentage of Negroes. It is due to the strong curved shaved hairs growing back into the skin and causing irritation, foreign body inflammation and eventually scarring. The clinical picture is characterized by hairs which extend for a short distance above and parallel to the surface, both ends being buried in the skin (loop hairs, *poils recourbés* of Dubreuilh, pili recurvi). The follicle of the hair is normal, while there is an inflammatory papule or papulopustule around the free end. Later in life, inflammatory reaction is less pronounced and the hairs lie in shallow grooves. Still later, the hairs degenerate, leaving the skin crisscrossed by narrow depressed scars, which give the cheeks of many of the older men a characteristic appearance. The designation of chronic scarring pseudofolliculitis (pseudofolliculitis chronica cicatrisans) is proposed for this condition.

Histologic examination by means of serial sections and three dimensional large scale reconstruction (Dr. Felix Pinkus) has shown that there is a fundamental difference between ingrown hairs of straight, wavy or curly beards and the loop hairs causing pseudofolliculitis. The ingrown hair of a white man is a hair which has been shaved too close, has slipped back beneath the surface and, owing to continuing growth, has made a false path through the side wall of the follicle into the cutis instead of emerging at the follicular opening. The negroid hair emerges normally but, owing to its inherent curve, grows back into the skin if it is not shaved close enough.

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DEMODEX FOLLICULORUM

ITS INCIDENCE IN ROUTINE HISTOLOGIC STUDY OF THE SKIN

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Demodex folliculorum has been suggested as a pathogen by several authors, particularly by Lawrence,[1] Ayres [2] and Ayres and Anderson.[3] Their views are not accepted without reservation by all dermatologists. These investigators admit that this arachnid occurs almost universally in small numbers in the hair follicles and sebaceous ducts of adult human beings and that under ordinary conditions it is apparently satisfied with the role of a scavenger. Since the mere factor of incidence of a micro-organism can scarcely determine whether or not it is pathogenic, the data to follow are not to be construed as pointing one way or the other.

The material utilized in this study consisted of 1,750 specimens of cutaneous tissue (obtained for biopsy or at necropsy) and routinely submitted for histologic diagnosis in this laboratory. In each instance the diagnosis of the mite was based on appearances which, once learned, are characteristic and not readily overlooked. In fact, after such self training, the microscopist learns to scrutinize subconsciously the pilosebaceous orifice for certain pink shreds and blue granules which he once overlooked, assuming they were strands of keratinous substance and nuclear debris. It is not claimed that any epoch-making results will accrue from an increasing recognition of this mite in histologic sections, but in any event the microscopist should be able to register anything and everything exhibited, whether of practical significance or not. This has not been the case in the past, and one of the aims of this communication is to call attention to the relatively common presence of the mite and to the fact that it is often overlooked.

Suspicion should be aroused as to D. folliculorum when in the pilosebaceous orifice certain aggregations of pink-staining material (hematoxylin and eosin stain) appear which are rather more sharply delimited than is keratinous debris. Such aggregations will be elongate or rounded, depending on the plane of section of the mite. Closer study may now disclose deeply staining, blue, spheroidal granules which are surrounded by a thin ring of pink cuticle.[4] Only the granules, which

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* Fellow in dermatology and syphilology in the University of Pennsylvania School of Medicine.

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1. Lawrence, H.: The Pathogenicity of the Demodex (Owen) in the Human Being, M. J. Australia **2**:39 (July 16) 1921; Further Observations upon the Pathogenicity of Demodex (Owen) in the Human Being, ibid. **2**:419, 1921; On a Skin Eruption Associated with the Presence of Great Numbers of Demodex Folliculorum, ibid. **2**:555, 1916. Lawrence, H., and Brodie, R.: Pityriasis Folliculorum (Demodex), ibid. **1**:529 (May 2) 1931. Lawrence, H.: Pathogenicity of Demodex Folliculorum in Man, Arch. Dermat. & Syph. **32**:633 (Oct.) 1935.

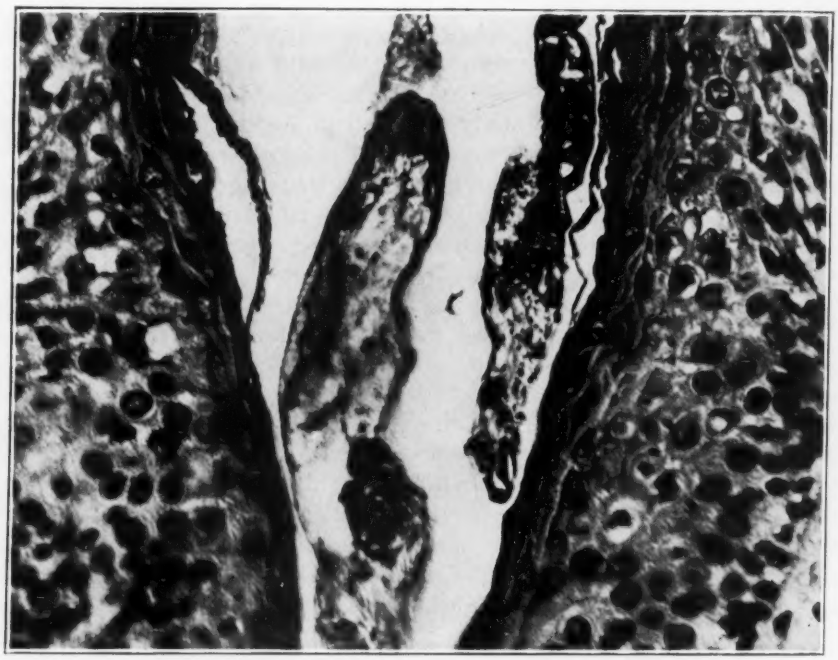
2. Ayres, S., Jr.: Pityriasis Folliculorum (Demodex), Arch. Dermat. & Syph. **21**:19 (Jan.) 1930.

3. Ayres, S., Jr., and Anderson, N. P.: Demodex Folliculorum: Its Role in the Etiology of Acne Rosacea, Arch. Dermat. & Syph. **25**:89 (Jan.) 1932.

4. Weidman, F. D., in discussion on Ayres and Anderson.[3]

represent some of the viscera of the parasite, are conspicuous in transverse sections. Perhaps the granules could be confused with Pityrosporum ovale; the former are smaller, however, and less regular in outline and staining qualities. The cuticle may resemble the loose keratinous material of the stratum corneum, but the stratum corneum never shows the transverse striations which appear so characteristic in D. folliculorum. This segmentation is not present on the capitulum; it is limited to the dorsum of the cephalothorax and extends over all surfaces of the abdomen and tail.[5] In short, a coral-like pink envelope around a cluster of blue granules calls for closer scrutiny for striations; this combination identifies D. folliculorum, at least as it is now known.

The use of such material in the form of sections immediately involves a number of objections, the most important of which follow: First, the diagnosis of the



Photomicrograph of the orifice of a hair follicle showing several specimens of Demodex. Note the segmentation of the cuticle, particularly in the upper portion of the large mite on the left. (Hematoxylin and eosin; × 440.)

presence or absence of the parasite was made on the examination of but a few (usually two to six) sections; rarely were serial sections employed. Accordingly, many of the parasites must have escaped detection, especially since D. folliculorum provokes no notable inflammatory response in the surrounding tissue to attract attention to it. In short, the incidence implied in this study is far too low. Second, specimens of skin submitted to the pathologist are most often of cancer, nevi and certain rare dermatoses which have challenged clinical diagnostic skill; therefore, they are not representative of clinical dermatology at large. In line with this discrepancy, statistically speaking, comes the admission that this series of 1,750 unselected cases included none of the "pityriasis folliculorum (demodex)" of Law-

5. Hirst, S.: Studies on Acari: I. The Genus Demodex, Owen, printed by order of the Trustees of the British Museum of Natural History, London, 1919.

rence and of Ayres, only 1 with the clinical diagnosis of demodecosis and 2 of rosacea. In the 2 cases of rosacea there was advanced rhinophyma. In spite of these shortcomings and several others that might be advanced, the following statistics may be of interest:

Number of specimens of skin examined			1,750
Number of specimens in which D. folliculorum was found			40 (2.28 per cent)
Histologic diagnosis:			
Nevic dermatoses		11	
Intraepidermal nevus	0		
Intradermal nevus	7		
Dermoepidermal nevus	0		
Adenoma sebaceum	4		
Epithelioma		17	
Basal cell	7		
Morphea-like basal cell	1		
Prickle cell	1		
Mixed	3		
Trichoepithelioma	4		
Sweat duct	1		
Nonmalignant and non-nevic miscellaneous dermatoses		12	

The only significant addition to this list is the additional diagnosis of hyperplasia of the sebaceous glands in 10 of the 40 instances, that is, 25 per cent. It was surprising that specimens from only 1 of the 2 cases of rhinophyma in the entire group contained the animal parasite, although several sections were examined critically.

The area of the body from which the material was obtained varied considerably, as is shown by the following tabulation of cases:

Head			34
Face		32	
Forehead	4		
Nose	9		
Cheek	7		
Temple	3		
Chin	3		
Eyebrow	2		
Eyelid	1		
Not specified	3		
Ear		2	
Pinna	1		
Auricle	1		
Breast			2
Shoulder			2
Back			1
Not specified			1
Total			40

The most frequent site of occurrence for the parasite, in approximately one fourth of the series, was the nose. Of the 9 cases, in 3 the material was from the nasal bridge and in the remaining 6 from the alae nasi.

Skin from the region of the breast was concerned in only 17 of the total 1,750 cases surveyed. The finding of Demodex in 2 gives an incidence of 8.5 per cent,

which is far short of the figures of Chambers and Somerset,[6] who reported that Demodex was found in the skin of the nipples of 60 per cent of the breasts amputated for carcinoma.

The ages of the patients in this series are apparently not important, since they ranged from 16 to 77 years in the 40 cases.

SUMMARY

In a survey of 1,750 consecutive samples of unselected tissue submitted for routine histologic study, Demodex folliculorum was encountered in 40. Of these, 17 were diagnosed as epithelioma and 7 as simple nevi; 32 of the 40 were from the face.

Dr. Fred D. Weidman gave advice and criticism and made available the material used in this survey.

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6. Chambers, H., and Somerset, A. M.: Breast Diseases and the Demodex Folliculorum, Lancet **1**:172, 1925.

DERMATITIS VENENATA CAUSED BY THE MANZANILLO TREE

FURTHER OBSERVATIONS AND REPORT OF SIXTY CASES

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One of us (E. M. S.) recently reported on the hazards of contact with Hippomane mancinella L., family Euphorbiaceae.[1] The common name for this poisonous tree is manzanillo; the names beach apple and shore apple are more recent ones.

It was pointed out that the departmental surgeon each year issues warnings to the military personnel stationed in the department from which the present observations are reported, relative to the dangers of this tree. Soldiers are repeatedly cautioned to avoid contact with it and to avoid the areas in which it grows.

We wish to submit a brief summary of 60 cases of dermatitis venenata caused by this tree, which we feel are of sufficient interest to be reported.

During spring maneuvers, a detachment of soldiers was given the problem of establishing a beach defense. The night was dark, and all the necessary positions and emplacements were established about midnight. About three hours later the first patient was brought to the first aid station and was seen by one of us (C. A. W.). His hands were held tightly over both eyes, and he was moaning because of severe pain. The soldier who accompanied this man stated that he had been awakened by the moans and restlessness of the patient and had brought him to the first aid station at once. The patient stated that he had been awakened by severe burning and pain of his face and that shortly after opening his eyes he noted that they too began to burn. These symptoms had increased in severity and at the time of examination he was in great distress. There were numerous erythematous macules of the face and neck, varying in size from that of a pencil head to that of a silver quarter; many were discrete, but many had become confluent and showed vesiculation. The conjunctivas were red and edematous. The edema became so severe that on the left side part of the palpebral conjunctiva overhung the lower lid. Examination was difficult because of strict black-out regulations. Morphine sulfate, ¼ grain (16 mg.), was given subcutaneously, warm saline compresses were applied to the face and neck and finally an ointment containing boric acid in 5 per cent concentration was applied before evacuating the patient to a hospital.

During the next four hours 60 men had to be evacuated because they were unfit for combat duty. Of this group at least 50 per cent were temporarily blinded because of severe conjunctivitis and edema of the eyelids. Many men had removed their shirts and trousers and slept rolled up in blankets; on arising they became contaminated on touching the tree or the surrounding wet sand. The affected areas were acutely erythematous and soon became edematous; in many of the men vesicles of various sizes developed, and in several bullas developed on the

1. Satulsky, E. M.: Dermatitis Venenata Caused by the Manzanillo Tree, Arch. Dermat. & Syph. **47**:36 (Jan.) 1943.

face and neck which varied in size from that of a golf ball to that of a crabapple. These lesions were opened aseptically, and warm saline compresses were applied. Morphine was required in the cases in which the conjunctivitis and the lesions of the lips were severe. All the patients were evacuated to a hospital, where the therapy consisted in application of saline compresses, saline irrigations of the eyes and local application of bland greases. All the patients recovered without sequelae, and most of them were returned to duty in seven to eight days.

Daylight revealed numerous manzanillo trees in the areas where the men had slept. The men had been repeatedly warned about the dangers of these trees but in the excitement and urgency of their duties many of them forgot the warnings. Questioning revealed that many of the men had not come into direct contact with the trees. The dew was particularly heavy that night, and from the distribution of the dermatologic lesions it may be assumed that the dew was the cause of the lesions. It has been noted [1] that the sap of this tree is so poisonous that the dew contains sufficient toxin to cause irritation and edema of the skin and mucous membranes.

As an effective treatment, immersion in salt water has been recommended by the departmental surgeon and has been used in the Panama Canal Department for years. Several of the men had gone into the ocean but had to leave immediately because of a fish known as the "sting ray" which inhabits the water in that vicinity.

It is interesting to note that 1 of the soldiers in the detachment was acquainted with the manzanillo tree and could handle the leaves, bark and fruit with impunity. He was also able to rub the milky latex into his skin without local reaction.

SUMMARY

A further report is made on the irritating action of the manzanillo (beach apple or shore apple) tree found in the Republic of Panama and the Canal Zone.

A brief summary of 60 cases of dermatitis venenata caused by this tree is reported.

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SENSITIVITY TO TUBERCULIN IN ACNE AND IN OTHER NONTUBERCULOUS DISEASES OF THE SKIN

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Ramel [1] found acid-fast bacilli in material taken from closed acne lesions of 4 persons in 1928 and of 10 person in 1930, none of whom presented clinical manifestations of tuberculosis. The organisms were verified as Mycobacterium tuberculosis by guinea pig inoculation. R. Griesbach,[2] also in 1930, observed acne to be a concomitant finding in more than 30 per cent of 18,000 cases of active pulmonary tuberculosis. Four years later von Kémeri [3] found roentgenographic evidence of pulmonary tuberculosis of moderate severity, positive reactions to tuberculin and foci of pyogenic infection in 52 persons with acne. Thirty were treated solely with injections of tuberculin administered in small, gradually increasing doses; in 27 the acne disappeared, in 2 it was improved and in 1 it became worse. Von Kémeri believed that acne resulted from a combined infection with tuberculosis and pyogenic organisms, the former being responsible for the chronicity of the disease and the latter for the pustular development. Franz von Veress [4] in 1938 investigated the temperature of 76 patients with acne and found it to be slightly elevated in 72. Van Studdiford,[5] in the same year, remarked on the difficulty of the classification of the various manifestations of acne and pointed out the resemblance of some of these to tuberculodermas of the face. He tested 49 patients with acne with tuberculin. Positive intracutaneous reactions were given by 29, 3 of whom presented tuberculous axillary adenitis, from which bovine tubercle

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1. Ramel, E.: (*a*) Ueber die Beziehungen der Tuberkulose zur Aetiologie der Acne vulgaris und Acne conglobata, Arch. f. Dermat. u. Syph. **155**:253, 1928; (*b*) Acné vulgaire et tuberculose, Schweiz. med. Wchnschr. **60**:754, 1930; (*c*) Le problème étiologique de l'acné vulgaire, Bull. Soc. franç. de dermat. et syph. **37**:1193, 1930.

2. Griesbach, R.: Hautkrankheiten bei Lungentuberkulose mit besonderer Berücksichtigung der Akne, Ztschr. f. Tuberk. **57**:405, 1930.

3. von Kémeri, D.: Aetiologie und allgemeine Behandlung der Akne vulgaris, Dermat. Wchnschr. **98**:237, 1934.

4. von Veress, F.: Akne und Tuberkulose, Dermat. Wchnschr. **107**:1519, 1938.

5. Van Studdiford, M. T.: Diagnostic and Therapeutic Use of Tuberculin in Certain Acneform Eruptions, Arch. Dermat. & Syph. **38**:737 (Nov.) 1938.

bacilli were isolated in 2. In 2 others bovine bacilli were found in lesions of the face. Twenty-one positive reactors were treated with intramuscular injections of tuberculin over a period of two months, with the result that 10 patients were cured and 11 showed improvement. Lichtenstein and Stillians,[6] however, treated 11 patients with acne with injections of tuberculin, without success. These authors noted that acne was by far the most frequent disease of the skin in the City of Chicago Municipal Tuberculosis Sanitarium and ascribed this to the average youthful age of the inmates. A large proportion of the inmates gave the history either of a clean skin before entry or of exacerbation of a preexisting acne within a few months after admission to the institution. Lichtenstein and Stillians expressed the belief that the fattening diet employed in the sanatorium was responsible for the initiation or exacerbation of the acne.

The summary of the relevant literature shows that no conclusion has yet been reached on the relation, if any, between acne and tuberculosis. The reports indicate that Myco. tuberculosis has been found in acne lesions by two investigators, that the cutaneous condition in a certain proportion of cases is benefited by injections of tuberculin, that acne is a common disease in tuberculosis sanatoriums, that a positive reaction to tuberculin occurs in a certain, but unknown, percentage of patients with acne and that tuberculous lesions of the lungs are found in patients with acne.

This paper offers the results of tuberculin tests[7] performed on a number of persons with and without acne.

MATERIALS AND METHODS

In the beginning of this study two varieties of tuberculin were employed concomitantly in each subject: old tuberculin, human (Lilly), and Mulford tuberculin, "OT" bovine type (Sharp & Dohme). The bovine type of tuberculin was employed because it has been stated[5] that persons with acne who react to this material may be anergic to the human type of tuberculin, and vice versa. In this study, in which 350 persons were tested with both materials, the reaction to the human was invariably stronger than that to the bovine tuberculin. At the commencement of this work the tests were performed with dilutions of 1 to 1,000,000, 1 to 100,000, 1 to 10,000 and 1 to 1,000. The use of the highest dilution, however, was soon abandoned, owing to the relatively few positive reactions obtained therewith. The dilutions were freshly prepared in sterile physi-

6. Lichtenstein, M. R., and Stillians, A. W.: (*a*) Tuberculin Treatment of Acne Vulgaris in Tuberculous Patients, Arch. Dermat. & Syph. **45**:956 (May) 1942; (*b*) Liver Extract in the Treatment of Acne Vulgaris in Tuberculous Patients, ibid. **45**:959 (May) 1942.

7. (*a*) Jadassohn, J.: Remarks on Tuberculin in Dermatology, Brit. J. Dermat. **41**:45, 1921. (*b*) Sulzberger, M. B., and Wise, F.: Tuberculin: Newer Dermatological Considerations and Reasons for Its More General Use in Diagnosis, M. Clin. North America **14**:1555, 1931.

ologic solution of sodium chloride immediately prior to use, and a separate syringe was employed for each dilution. Tests were performed on the flexor surfaces of the forearms, all dilutions of both tuberculins being used at one session. Reactions were read forty-eight hours after the performance of the test and subsequently at frequent intervals. The responses were graded as follows: 4 plus, edema and erythema measuring more than 20 mm. in diameter with necrosis; 3 plus, edema and erythema measuring more than 20 mm. without necrosis; 2 plus, edema and erythema measuring from 10 to 20 mm.; 1 plus, edema and erythema measuring less than 10 mm. in diameter. The patch test with tuberculin [8] was also employed, generally in conjunction with the intradermal but occasionally as the only test. Material for the performance of the patch test was obtained from the Lederle Laboratories and consisted of three filter paper squares, two of which were saturated with tuberculin while the third served as a control. The squares were placed on a strip of adhesive tape, which was applied to the middle of the chest, allowed to remain in position for forty-eight hours and then removed. A positive reaction usually appeared as a lichenoid follicular or vesicular follicular eruption sharply limited to the area of the test. Occasionally a full development of the reaction was delayed for seventy-two hours or longer after the removal of the material. Quantitative studies were not performed with the patch test; the material was obtainable only in a single concentration of tuberculin. There is, however, close agreement in about 90 and more per cent of cases between the result of the intradermal test with 0.1 mg. of old tuberculin and that of the patch test.[9]

The group of patients under investigation from September 1940 until June 1942 consisted of 231 persons, of whom 102 had acne and 129 presented other cutaneous diseases. The latter were employed as controls. All were in the lower economic brackets. The diagnosis of acne rested on the presence of a combination of the following signs: seborrhea, comedos, follicular papules and pustules, cystic lesions and scarring, occurring on the face, chest and back. Biopsies were not performed. No patient was included in the test group if there was any doubt in the diagnosis of acne; similarly, no person was admitted to the control group who was known to have tuberculosis of the lungs, a tuberculous infection of the skin or an eruption of even questionable tuberculous nature, such as lupus erythematosus, granuloma annulare or erythema induratum. No patient was seen to present both acne and cutaneous tuberculosis.

The subjects were divided into two age groups, namely, from 13 through 25 years and from 26 through 40. This age classification was chosen because acne is seldom seen before 13 years or after 40 and begins to decline in frequency after the age of 25.

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8. Vollmer, H., and Goldberger, E. W.: (*a*) A New Tuberculin Patch Test, Am. J. Dis. Child. **54**:1019 (Nov.) 1937; (*b*) Comparative Study of the Tuberculin Patch Test and the Mantoux Intracutaneous Tests, ibid. **56**:584 (Sept.) 1938.

9. (*a*) Steward, W. D.: The New Tuberculin Patch Test, J. Pediat. **13**:510, 1938. (*b*) Weiner, S. B., and Neustadt, A.: Tuberculin Patch Test: A Comparison with the Pirquet and Mantoux Test, ibid. **14**:752, 1939. (*c*) Vollmer, H., and Goldberger, E. W.: Evaluation of the Tuberculin Patch Test (Vollmer-Lederle), Am. J. Dis. Child. **57**:1272 (June) 1939. (*d*) Taylor, G.: Tuberculin Patch Test: A Comparison with the Mantoux Intracutaneous Test, Am. Rev. Tuberc. **40**:236, 1939.

RESULTS

The results of the tests are shown in the accompanying table.

The proportion of positive reactors in the younger age group was practically the same among the control subjects and the patients with acne. With the older patients there was a considerably smaller proportion of positive reactions in those with acne than in the controls. The comparative results in the older age group may have been influenced by the relatively small number of patients with acne tested. Should, however, the consideration of a larger series of such patients over the age of 25 support the findings presented in this paper, it must be concluded that older persons with acne are distinctly hypoergic to intradermal or patch tests with tuberculin.

The material was further analyzed to determine the relation, if any, between the severity of the acne eruption and the intensity of the response to tuberculin. In 12 of 16 cases of severe acne the reactions

Results of Intradermal and Patch Tests with Tuberculin in Patients with Acne and in Control Subjects

	1:100,000 Dilution				1:1,000 Dilution			
	Control Subjects		Patients with Acne		Control Subjects		Patients with Acne	
Age Group, Years	Number	Per Cent Positive Reactions	Number	Per Cent Positive Reactions	Number	Per Cent Positive Reactions	Number	Per Cent Positive Reactions
13 through 25	56	7.1	77	7.8	65	37.0	87	33.3
26 through 40	53	22.6	14	7.1	64	82.8	15	46.7

varied from negative to weakly positive, and 10 of 15 strong reactions were associated with mild acne. It would appear, therefore, that a severe degree of acne is likely to give a weak reaction and a moderate degree of acne a strong cutaneous response to tuberculin.

SUMMARY AND CONCLUSIONS

The relevant literature shows that no conclusion has been reached as yet as to a possible relation, if any, between acne and tuberculosis.

The present paper offers a comparison of the sensitivity to tuberculin of 102 persons with acne and 129 persons with other nontuberculous cutaneous diseases, the latter serving as controls.

While in the age group from 13 through 25 years the proportion of positive reactors among the subjects with acne and the controls was practically the same, in the age group from 26 through 40 years there was a decidedly smaller proportion of positive reactors among those with acne than among the controls.

A further analysis of the material showed that patients with severe acne tended to show negative to weakly positive reactions to tuberculin and that strongly positive reactions were associated with mild acne.

There appears to be a relation between the degree of sensitivity of the skin to tuberculin and (*a*) acne as it occurs in older age groups or (*b*) the severity of the acne. The nature of the relation, however, requires further elucidation.

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LENTIGINES: THEIR POSSIBLE SIGNIFICANCE

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Despite the fact that most textbooks [1] make no distinction between lentigines and freckles (ephelides), listing them as synonymous, many observers [2] have noted distinct differences between them. Besides listing such differences, it is the purpose of this paper to show both age and seasonal variations in the number of lentigines and to discuss their possible clinical significance.

Lentigines are brown-pigmented lesions, darker than freckles, flat or raised, seen on all parts of the skin and present in almost every child and adult. They are absent at birth, usually appear about the second year of life and increase in number with age. They were so numerous on children with frequent colds in the pediatric clinic for diseases of the chest at the New York Post-Graduate Medical School and Hospital that a survey was made covering a three year period, and variations in their numbers were recorded at intervals. The counts of lentigines

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1. (*a*) Thomson, A. T.: Diseases Affecting the Skin, London, Longman Brown, Green and Longmans, 1850, p. 396. (*b*) Duhring, L. A.: Diseases of the Skin, Philadelphia, J. B. Lippincott & Co., 1882, p. 368. (*c*) Shoemaker, J. V.: Diseases of the Skin, New York, D. Appleton and Company, 1888, p. 340. (*d*) Van Harlingen, A.: Skin Diseases, ed. 3, Philadelphia, P. Blakiston, Son & Co., 1895, p. 385. (*e*) Hardaway, W. A.: Skin Diseases, Philadelphia, Lea Brothers & Co., 1898, p. 243. (*f*) Fox, G. H.: Photographic Atlas of the Diseases of the Skin, Philadelphia, J. B. Lippincott Co., 1905, vol. 2, p. 119. (*g*) Jackson, G. T.: Diseases of the Skin, ed. 6, New York, Lea & Febiger, 1908, p. 391. (*h*) Hyde, J. N.: Diseases of the Skin, New York, Lea & Febiger, 1909, p. 531. (*i*) Dearborn, F. M.: Diseases of the Skin, Including the Exanthemata, Philadelphia, Boericke & Tafel, 1913, p. 273. (*j*) Morris, M.: Diseases of the Skin, ed. 6, New York, William Wood & Company, 1917. (*k*) Macleod, J. M. H.: Diseases of the Skin, London, H. K. Lewis & Co., Ltd., 1920. (*l*) Highman, W. J.: Dermatology, New York, The Macmillan Company, 1921. (*m*) Schamberg, F. F.: Diseases of the Skin and the Eruptive Fevers, Philadelphia, W. B. Saunders Company, 1921, p. 230. (*n*) Stelwagon, H. W.: Diseases of the Skin, ibid., 1923, p. 521. (*o*) Pusey, W. A.: Principles and Practice of Dermatology, ed. 4, New York, D. Appleton and Company, 1925, p. 965. (*p*) Walker, N.: Introduction to Dermatology, ed. 8, Edinburgh, W. Green & Son., Ltd., 1925, p. 359. (*q*) Sequeira, J. H.: Diseases of the Skin, ed. 4, New York, The Macmillan Company, 1927. (*r*) Strickler, A.: Diseases of the Skin and Syphilis, Philadelphia, F. A. Davis Company, 1927, p. 587. (*s*) Fox, H.: Diseases of the Skin in Infancy and Childhood, New York, D. Appleton and Company, 1930. (*t*) Ormsby, O. S.: Diseases of the Skin, ed. 4. Philadelphia, Lea & Febiger, 1934, p. 587, (*u*) Knowles, F, C.: Diseases of the Skin, ed. 3, ibid., 1935, p. 273. (*v*) MacKee, G. M., and Cippolaro, A. C.: Skin Diseases in Children, New York, Paul B. Hoeber, Inc., 1936, p. 250. (*w*) Andrews, G. C.: Diseases of the Skin, ed. 2, Philadelphia, W. B. Saunders Company, 1938, p. 84. (*x*) Sutton, R. L., and Sutton, R. L., Jr.: Diseases of the Skin, ed. 10, St. Louis, C. V. Mosby Company, 1939, p. 542. (*y*) Walker, N., and Percival, G. H.: Dermatology, ed. 10, Baltimore, Williams & Wilkins Company, 1939, p. 381. (*z*) Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

2. (*a*) Wilson, E.: Diseases of the Skin, ed. 6, Philadelphia, Lea & Blanchard, 1865, pp. 537 and 538. (*b*) Hebra, F., and Kaposi, M.: Diseases of the Skin, Including the Exanthemata, translated and edited by W. Tay, London, The New Sydenham Society, 1874, pp. 2, 5 and 7. (*c*) Celsus, cited by Hebra and Kaposi,[2b] p. 2. (*d*) Lorry, cited by Hebra and Kaposi,[2b] p. 2. (*e*) Lesser, E., in von Ziemssen, H.: Handbook of Diseases of the Skin, New York, William Wood & Company, 1885, p. 440. (*f*) Kaposi, M.: Pathology and Treatment of Diseases of the Skin, translated under supervision of J. C. Johnston, New York,

(Footnote continued on next page)

on these children were compared with counts on children in other departments of the hospital, as well as with counts on children and adults in private practice. Altogether, lentigines were counted on a total of 850 patients.

In this study nevi spili and moles were recorded as lentigines. Nevi spili are sometimes described as flat pigmented moles and are differentiated by some observers from lentigines. However, it is probable that the latter start as nevi spili, which represent an early transition stage.[3] When lentigines become papular or raised, they are commonly referred to as moles.

There are many differences between lentigines and freckles (table 1).

The large number of blemishes (lentigines, moles) appearing on children visiting the pediatric clinic for diseases of the chest, with complaints of frequent colds, recurring bronchitis, bronchiectasis and primary tuberculous infection, was in contrast to the relatively small number seen on healthy children and the complete absence of such blemishes on newborn infants. To confirm this impression, control groups were sought and comparisons made.

Four comparatively healthy control groups consisted of the following: (1) patients in the general clinic; (2) children in private practice who were seldom ill; (3) newborn infants seen at Queens General Hospital; (4) adolescents and adults met in private practice in whom colds were infrequent and who were free of constitutional disease. The number of control children and adults having few colds or infections was small when judged strictly (tables 2 and 3).

The chronically or recurrently infected groups were children in the pediatric clinic for diseases of the chest, the clinic for patients with allergy,[4] the clinic for cardiac research[5] (rheumatic children), and adults seen privately who frequently had colds and infections of a constitutional nature.

PROCEDURE

Lentigines were counted on five areas in each patient as follows: (1) left upper extremity; (2) right upper extremity; (3) chest; (4) back; (5) face, including neck. The counts on these five areas in each patient were totaled.

Counts on the lower part of the trunk and on the lower extremities were in no instance tabulated, although it was observed in several cases that the average number in these areas was considerably smaller, as noted by Pack and Livingston,[2r] being approximately 10 per cent of the total in the five areas listed. This explanation is made in order that the total count of lentigines recorded here may be compared with the totals noted by others (table 1, factor 7).

William Wood & Company, 1895, pp. 415 and 418. (*g*) Saalfeld, E.: Lectures on Cosmetic Treatment, translated by J. F. H. Dally, New York, Paul B. Hoeber, 1910, p. 108. (*h*) Sabouraud, R.: Regional Topographical Dermatology, translated by C. F. Marshall, New York, Rebman Co., 1913, pp. 6 and 612. (*i*) Ahlswede, E.: Practical Treatment of Skin Diseases, New York, Paul B. Hoeber, Inc., 1932, p. 373. (*j*) Meirowsky, E., in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1933, vol. 4, pt. 2, pp. 652-655, 708-716 and 1022. (*k*) Gougerot, Meyer and Uhry, cited by Meirowsky.[2j] (*l*) Gougerot, cited by Meirowsky.[2j] (*m*) Darier; Balzer; Gaucher and Milian; Audry; Kiess, and Almkvist, cited by Meirowsky.[2j] (*n*) Darier, cited by Meirowsky.[2j] (*o*) Pollio, cited by Meirowsky.[2j] (*p*) Jadassohn, cited by Meirowsky.[2j] (*q*) Michel, cited by Meirowsky.[2j] (*r*) Pack, G. T., and Livingston, E. M.: The Treatment of Pigmented Nevi and Melanomas, in Treatment of Cancer and Allied Diseases, New York, Paul B. Hoeber, Inc., 1940, p. 2072. (*s*) Siemens, cited by Meirowsky.[2j] (*t*) Macleod, J. M. H., and Muende, I.: Pathology of the Skin, ed. 2, New York, Paul B. Hoeber, Inc., 1940, p. 320. (*u*) Becker, S. W., and Obermeyer, M. E.: Modern Dermatology and Syphilology, Philadelphia, J. B. Lippincott Company, 1940, p. 293. (*v*) Eller, J. J.: Tumors of the Skin (Benign and Malignant), Philadelphia, Lea & Febiger, 1939, p. 71. (*w*) Goldsmith, W. N.: Recent Advances in Dermatology, Philadelphia, P. Blakiston's Son & Co., 1936, pp. 44 and 46. (*x*) Traub, E. F., and Keil, H.: The "Common Mole," Arch. Dermat. & Syph. **41**:214 (Feb.) 1940.

3. Meirowsky.[2j] Eller.[2v]

4. The counts were made with the permission of Dr. Robert Chobot, who is in charge.

5. The counts were made with the permission of Dr. Valentina P. Wasson, who is in charge.

TABLE 1.—*Differences Between Lentigines and Freckles (Ephelides)*

	Lentigines	Freckles
1. Age of onset	Early At 2 years [2f]	Later 6 to 8 years [2e,f]
2. Season of onset	Any season [2a,c,d]	Summer
3. Increase in number following exposure to sunlight	No	Yes
4. Conspicuousness: (a) in summer	Yes, except that small ones fade	Yes
(b) in winter	Yes	No
4. Location on body	All parts; discretely distributed [2f,g,h]	Nose, cheeks, forehead; less commonly arms, legs, back, chest
6. Incidence in: (a) fair-skinned	Present	Many present [2g]
(b) brunettes	Present	Absent or few [2e]
(c) mulattoes	Present	Many present [1w]
(d) Negroes	Present in small numbers	Absent
(e) Japanese	Present	Absent or extremely rare (Adachi, cited by Goldsmith [2w])
(f) general incidence	90-100% (Cannon, A. B.: New York State J. Med. **40**: 1567 [Nov. 1] 1940. Nicholson, G. W.: Guy's Hosp. Rep. **71**: 222 [April] 1921; **72**: 193 [April] and 333 [July] 1922; **73**: 37 [Jan.], 164 [April] and 298 [July] 1923. Siemens [2s]	As noted above
7. Number when present	Few (1 to 150); av. per person 10 to 30 [2q,r,s]; (see also table 2)	Several to thousands
8. Occurrence as single lesion	Yes [2e,f,v]	No [2e]
9. Coalescence of lesions when numerous	No, except in Addison's disease [2e]	Yes [1w]
10. Size of lesions	Variable Pinhead to lentil size [2b] or larger [2f,u]	Not so variable Pinhead to pea size [2v]
11. Increase in size of lesions	Yes [2f,i,q,u]	No, or slight
12. Color	Darker [2e]	Lighter [2e]; unevenly tinged [2f]
13. Extension of pigment to connective tissue	Yes [2i,v]	No [2c]
14. Pigment-producing, dopa-positive reaction	Yes (Bloch, B.: Ztschr. f. physiol. Chem. **98**: 226, 1917. Macleod and Muende.[2t] Goldsmith.[2w])	No
15. Shape	Usually round, may be lenticular	Irregular (jagged, serrated); may be round
16. Sharp demarcation	Yes [2f]	Yes, but often less distinctly
17. Flat or raised	Flat at onset, becoming raised (papular)	Flat (macular)
18. Development of hair over lesions	Yes [2e]	No
19. Development into other forms	Yes [2f,j,p,q,v,w]	No
20. Development into cancer	Yes (Gartner, S.: Am. J. Ophth. **23**: 73 [Jan.] 1940. Kaposi.[2f] Pack and Livingston.[2r]); rare (lentigo maligna)	No
21. Permanence	Yes [2a,f,g]; exceptions noted in text	No
22. Presence in old age	Yes, although devolution is usual	No [2e,f,g]
23. Devolution (retrograde development, atrophy or disappearance)	Yes [2m,w,x]; not complete in larger lesions	Yes; complete in advanced age [2f]
24. Presence of nevus cells in lesions	Yes [2n,o,v,w]	No
25. Association with constitutional factors	Yes	No
26. Association with Addison's disease, von Recklinghausen's disease, Still's disease	Yes	No
27. Noted following typhoid fever, diphtheria, tuberculosis	Yes	No
28. Related to hypoadrenia	Yes	No
29. Hereditary tendency	Slight [2s]	Great [2w]
30. Resistance to treatment	Greater [2i]	

In the pediatric clinic for diseases of the chest 88 children were observed. Among the sickest were 22 children treated for active chronic purulent sinusitis or bronchosinusitis with autogenous vaccine made from cultures of secretions of their sinuses. On these 22 children counts were made periodically for three years. At first they were made bimonthly; later, at least two counts were taken per year, in the winter-spring and in the autumn. When it was noted that lentigines were generally most numerous in the spring and fewest in the fall, counts at these two seasons were used for purposes of comparison in this study.

In the clinic for patients with allergy, lentigines were counted on 69 children, most of whom were suffering from hay fever, vasomotor rhinitis and asthma. Almost all of these children had complicating sinusitis.

TABLE 2.—*Number of Lentigines Among Eight Hundred and Fifty Patients*

	Number of Patients	Average Age (Yr.)	Average Number of Lentigines
Children in clinic for allergic patients	69	8.6	28.6
Rheumatic children	159	11.6	25.0
Children in clinic for children with diseases of the chest: Total	88	8.9	24.6
Children with bronchiectasis	6	9.2	30.3
Children in general clinic: Total	72	8.1	15.5
Children with frequent colds *	57	7.8	16.5
Children with infrequent colds	9	9.2	12.3
Children with rare colds	6	9.6	3.6
Children in private practice: Total	159	8.1	25.4
Children with frequent colds *	120	8.4	30.4
Children with infrequent colds	32	7.1	10.9
Children with rare colds	7	9.0	9.3
Newborn infants (Queens General Hospital)	60	0.0	0.0
Adolescent children in private practice	31	17 to 20	30.2
Children with frequent colds *	25		32.8
Children with infrequent colds	6		19.3
Adults in private practice: Total	212	21 and older	21.0
Adults with frequent colds.*	141		24.5
Adults with infrequent colds	62		14.8
Adults with rare colds	9		7.9

* The term colds as used includes constitutional infections, as explained in the text.

TABLE 3.—*Number of Lentigines in Adults by Age Groups*

	Number of Patients	Average Number of Lentigines
Adults 21 to 45 years old: Total	143	23.0
Adults with frequent colds *	100	26.2
Adults with infrequent colds	37	17.0
Adults with rare colds	6	9.5
Adults 46 to 60 years old: Total	44	19.6
Adults with frequent colds *	29	23.6
Adults with infrequent colds	15	11.9
Adults 61 years of age and older: Total	25	11.2
Adults with frequent colds *	12	12.8
Adults with infrequent colds	13	9.8

* The term colds as used includes constitutional infections, as explained in the text.

In the clinic for cardiac research, counts were made on 159 rheumatic children; seasonal variations were noted during a two year period.

In the general clinic counts were made on 72 children, in whom the general health level was higher than in the preceding groups. However, many of these children had frequent colds and sinusitis, so that they were further subdivided into those susceptible to colds and those free of colds. The children in the cold-susceptible group were generally not as sick, nor did they have colds quite as frequently, as those sent to the clinic for children with diseases of the chest. These statements explain why the children of the general clinic were placed in our first control group of "comparatively healthy" children.

In private practice counts were made on 159 children. It so happened that many of these were seen for frequent colds and bronchial conditions comparable to those in the pediatric clinic for diseases of the chest. These children were also subdivided into a cold-susceptible and a cold-free group, the latter representing the second control group.

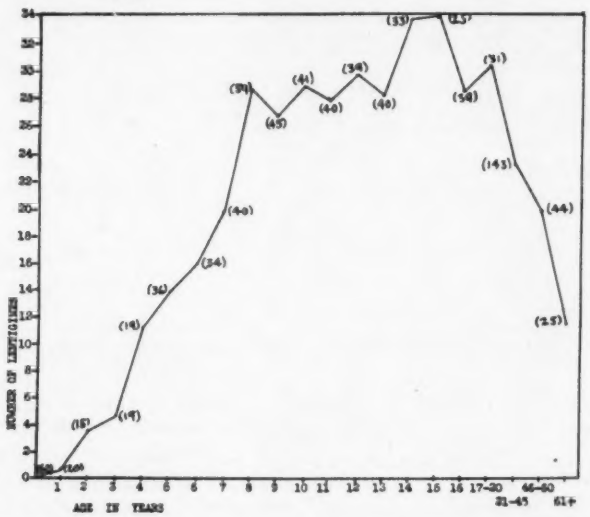
In private practice 31 adolescent youths, aged 17 to 20, inclusive, were observed, as well as a total of 212 adults. These were subdivided clinically into a "chronically infected" and a "comparatively healthy" group, the latter forming a third control group. In the chronically infected group were included those with active chronic sinusitis, recurring rheumatic pains, arthritis, recurring bronchitis and asthma, chronic colitis, diabetes and other chronic ailments. Among the controls were those who had no occasion to consult a physician for years, who may have come to the physician because of an accident, a furuncle or some nonconstitutional disease and whose history revealed no chronic ailment.

Sixty newborn babies constituted a fourth control group.

The counts of lentigines for our various groups of patients are given in table 2.

RESULTS

Variations in Number and Form of Lentigines with Age.—The 60 newborn infants were all free of lentigines. The development of these lesions was noted rarely in the first year, occasionally in the second year and most commonly in the third year or later, appearing on all parts of the body. In contrast, freckles were seldom observed before the fourth year and usually appeared first on the nose and adjacent areas.



Incidence of lentigines through life. The numbers on the curve represent numbers of cases.

As seen in the accompanying chart, lentigines increase in number with age up to about 20 years, developing rapidly in the ninth and tenth years. At age 8 the average number is 28. At ages 9, 10, 11, 12 and 13 the count remains about the same. At ages 14 and 15 the number of lentigines increases to 34, and at age 16 the count is again 28. In 31 adolescents, whose ages ranged from 17 through 20, the count increased slightly to 30. The count then drops in adults, 21 to 45 years old, to an average of 23, dropping again in the 46 to 60 year old ones to 20, and dropping still further in the group 61 years of age and older to only 11 lentigines (chart and table 3).

These figures are not in accord with those of Michel,[2q] who found an average of only 10 lentigines among 1,000 persons. He stated that the greatest number of single nevi developed approximately at the age of 10 to 15 years, and that the maximum total count was reached between 30 and 40 years of age. Siemens,[2s] on the other hand, found an average of 30 lentigines per person, three times Michel's count. An intermediate count, 18 lentigines per person, was reported by Pack and Livingston.[2r]

Changes in the form of the lesions were frequently observed in adults. It was at first hard to understand the apparent disappearance of lentigines in middle age and beyond (as noted by their diminishing number) and the appearance of other lesions, some similar to but others different from those in the younger groups. A review of the literature explained these transformations.

The most common change is depigmentation of the papular lentigo (mole); this results in a papilloma. Later in life, if the papilloma is large, and subcutaneous atrophy takes place, the resulting lesion is molluscum fibrosum, the lesion frequently found late in von Recklinghausen's disease. If the pigment, melanin, remains in the larger moles, but irregularities are noted on the surface, with sclerosis and hypertrophy of the epidermal elements, the resulting lesion is nevus verrucosus or warty mole. If this lesion becomes depigmented, the term molluscum verrucosum can be applied. Thus the larger lesions remain but frequently change their form.

The decrease in the number of lentigines in old age is apparently due to depigmentation of the smaller lesions. It is the smaller lesions, also, which seem to disappear during the summer and fall, thus accounting for the seasonal decrease in the warm months, about to be discussed.

Variations in Number of Lentigines with Season.—Seasonal variations in the number of lentigines were seen in 22 children with frequent colds followed for three years. The semiannual average counts (winter-spring and fall) for these 22 children, beginning in the winter-spring of 1938, are as follows: 24, 23; 24.8, 20.8; 37.2 and 34.9. The last count is for the fall of 1940. Thus the average increase in number of lentigines in two years, from the spring of 1938 to the spring of 1940, is 13.2, and that from the fall of 1938 to the fall of 1940 is 11.9, at an average age increase from 8.5 to 10.5 years. These were 22 sick children. In contrast other groups at this age whose health was better showed lesser seasonal and age fluctuations.

The change in number of lentigines is more consistent from fall to spring than from spring to fall, the trend to increase in number by spring being more regular. Among the 22 children 15 (70 per cent) showed an increase from fall 1938 to spring 1939. In the following year, also from fall to spring, 18 patients (82 per cent) showed an increase. Some of these increases in lentigo formation are phenomenal; 5 children showed a total rise of exactly 200 lentigines, an average increase of 40 per child.

The general trend from spring to fall is a decrease in the number of lentigines, but exceptions were noted, especially when infection continued during the summer. A good example is a child who had 61 lentigines in the spring of 1938 and 91 in the fall. He had three prolonged colds (allergic?) during that summer. Nevertheless, a decrease in number of lentigines during the summers of the three years was noted in more than half of the children.

The winter-spring increase in the total number of lentigines is considerably greater than the fall decrease. In my 22 patients the total increase in the number of lentigines during the winter-spring of 1939 and of 1940 over the previous fall count was 39 and 361, respectively. The total decrease of lentigines through the summer of 1938, of 1939 and of 1940 was, respectively, 22, 87 and 52. The possible significance of the great increase of lentigines during the winter of 1940 (361 lentigines, or 16.4 per patient) is discussed at the end of the article.

Among rheumatic children the seasonal variations followed the same pattern as in children of the pediatric clinic for diseases of the chest, the only difference being the lack of increase with age between 8 and 13, the age trend in the rheumatic group conforming to that in all children as shown in the chart. The average

count for 53 rheumatic children in the fall of 1940 was 28. In the following spring (1941) the same children had an average of 35, an increase of 7 lentigines. There were 37 increases and 12 decreases; 4 counts remained unchanged. In the spring of 1940, 40 children had an average of 30 lentigines. The count by fall dropped to 25. In this group 12 showed an increase and 23 a decrease, 5 remaining unchanged. In the fall of 1941 the average count among 44 children was 26; by the following spring (1942) the count increased to 36, an average increase of 10 lentigines per patient. In this group, 41 children showed an increase, 2 a decrease, and one count remained unchanged.

Controls.—In the control groups (the "comparatively healthy") I found few lentigines; none at all in newborn infants. In the general clinic the average count for 72 patients at the age of 8.1 years was 15.5 lentigines, in contrast to an average count of 25.7 for the rheumatic and the allergic groups and the children of the clinic for diseases of the chest combined (316 patients at an average age of 10.3 years). When only those children who gave a history of rare colds or infections were considered, a greater disparity was observed: Among 13 such children at an average age of 9.3 years the count was only 6.7.

Among adults with infrequent colds and constitutional infections a considerable disproportion of lentigines was noted when the count for this group was contrasted with the count for the sicker group (table 3).

COMMENT

There are four main aspects to a complete discussion of lentigo: First, the transitions of the lesion, including the corresponding nomenclature; second, the variations with age; third, the seasonal variations; fourth the significance of the lesion.

To one reviewing the literature it becomes obvious that various confusing terms are used to describe both lentigo and ephelis, as well as lesions which apparently represent further developments of lentigo. It is not pertinent here to discuss this conflict of nomenclature or to go into a detailed discussion of the transitions which take place in lentigo at various ages.

It is more pertinent to ask, Why does lentigo form?

Certainly, lentigines are not dependent on sunlight. In their comparatively small numbers they can scarcely be expected to protect the individual against actinic rays as freckles do. Furthermore, appearing in dark-skinned as well as in light-complexioned persons, unlike freckles, it would seem unreasonable to assume that they are intended by nature to protect against sunlight. Finally, lentigines appear in the winter and on parts unexposed to sunshine.

Further discussion of the origin of lentigo embraces the following considerations: What are the circumstances which precede the formation of the lentigo? What is the significance of the seasonal variations noted? Why does the incidence increase with age? Why is there a discrepancy of number counted in the various groups studied?

Certain clinical observations bear on these questions, notably the relation of lentigo to infection, to intensity of infection and to hypoadrenia.

Relation to Infections.—When lentigines appear early in life or in profusion, it is usually found that infection preceded their appearance. For example, a child aged 2, with pertussis of two months' duration, contracted bronchopneumonia and was carefully watched for lentigines; 7 small lesions developed on the chest and the back during the month of bronchopneumonia. Another child aged 2, with repeated purulent otitis and pronounced anemia, had 11 lentigines before his second birthday. Other examples were observed. In contrast, among 20

healthy children who had only minor colds, lentigines averaged 0.6 at the age of 1 year and 2.0 at the age of 2 years. Mothers who had become lentigo conscious during the period of observation of their children called attention to the development of new "spots" following a prolonged cold.

Infections of many kinds have been reported as preceding the onset of lentigines. They include: chorea,[6] diphtheria,[2e] typhoid fever,[2k] malaria,[7] "frequent attacks of sore throat with high fever," [8] chronic progressive generalized infantile arthritis,[9] rheumatoid arthritis [10] and poliomyelitis.[11] In poliomyelitis, Draper and Dupertuis [11] stated, "The importance of pigment irregularities, especially in the form of small black spots, is definitely the most outstanding and obscure feature." They reported 94 per cent of affected boys with "black spots" as against 47 per cent of well boys.

Many other diseases, the causes of which are still obscure but the clinical aspects of which are suggestive of infection, are complicated by lentigines or brownish pigmentations. Such diseases are pernicious anemia,[2j], von Recklinghausen's disease,[12] Still's disease [13] and Addison's disease.[14] Meirowsky [2j] emphasized that in Addison's disease acute infection commonly precedes the pigmentation.

The interval of time between the infection and the pigmentation varies. Within one month 7 lentigines developed in a child with bronchopneumonia following pertussis. Valenti [7a] described a case of malaria with Addison's disease in which lentigines developed within twenty-five days. In a 9 year old child pigmented spots developed two and a quarter months after a cold in the head and chorea, progressing into the full-blown pigmentation of Addison's disease.[6]

In some cases the pigmented lesions precede, rather than follow, the full-blown manifestations of infection. Thus Husler [9] in regard to chronic progressive generalized infantile arthritis stated:

> . . . The pigmentations which accompany the disease and which are emphasized by many observers are remarkable. In the prodrome, two thirds of the cases show marked ephelides; then, in progressive cachexia, intensive, diffuse brownish pigmentations are found at the sides of the forehead and temples. . . . The pigmentations are not the result of any exogenous therapeutic irradiation.

Even in the lentigo itself there is evidence of infection, an inflammatory reaction being found in the cutis [2x]

Relation to Intensity of Infection.—When infections are continuous, intense and prolonged, lentigines are both more numerous and increasingly pigmented, hypertrophying to the papular or molar stage. This was observed especially in

6. Snelling, C. E., and Erb, I. H.: Suprarenal Atrophy, J. Pediat. **7**:669 (Nov.) 1935.

7. (*a*) Valenti, F.: Sintomi del morbo di Addison nella Malaria, Policlinico (sez. prat.) **14**:1518, 1907. (*b*) de Brun, H.: Étude sur le pneumo-paludisme du sommet, Rev. de méd., Paris **15**:929, 1895.

8. Leader, S. D., and Grand, M. J. H.: Von Recklinghausen's Disease in Children: Report of a Case Presenting Cutaneous Pigmentation and Bone Changes, J. Pediat. **1**:754 (Dec.) 1932.

9. Husler, J., in von Pfaundler, M., and Schlossmann, A.: The Diseases of Children, Philadelphia, J. B. Lippincott Company, 1935, vol. 5, p. 430.

10. French, H.: An Index of Differential Diagnosis of Main Symptoms, ed. 5, Baltimore, William Wood & Company, 1936, p. 419.

11. Draper, G., and Dupertuis, C. W.: The Nature of the Human Factor in Infantile Paralysis: II. Relation of Age to Maturing Achievement and the Disease Picture, J. Clin. Investigation **18**:95 (Jan.) 1939.

12. Wickham, L.: Paris Letter, Brit. J. Dermat. **2**:151, 1890. Meirowsky.[2j] French.[10]

13. Leichtentritt, B., in Pfaundler, M., and Schlossmann, A.: The Diseases of Children, Philadelphia, J. B. Lippincott Company, 1935, vol. 3, p. 374.

14. Osler, W.: The Principles and Practice of Medicine, ed. 13, New York, D. Appleton-Century Co., 1938, p. 1107. Rowntree, L. G.: Increasing Survival Time in Addison's Disease, J. A. M. A. **114**:2526 (June 29) 1940.

6 patients with bronchiectasis, 5 of whom had at least 1 mole or papular lentigo. They averaged 30.3 lentigines, a high number, at an average age of 9.2 years. In a case of bronchiectasis reported by Ramond [15] the pigment became so profuse that he labeled it addisonian melanoderma.

Other groups with numerous lentigines included those 22 children in the pediatric clinic for diseases of the chest whose sinus infection was constant and troublesome; at the age of 10.5 years they averaged 37 lentigines, the greatest number of any group examined. Many of these children exhibited moles. Asthmatic children showed numerous lentigines and mole formation; this was particularly true of the children with infectious or perennial asthmatic conditions and chronically infected sinuses. The rheumatic children showed a great number of lentigines; 100 per cent of these children were reported to have chronic suppurative sinusitis,[16] with other possible foci limited.

Relation to Hypoadrenia.—Hypoadrenia may be said to occur in allergy, in Addison's disease and after severe infections that damage the adrenal glands.

If one agrees with Garretson,[17] J. H. Burn, J. A. Haiman and others that allergy is hypoadrenia, as judged by the accompanying hypotension and response to epinephrine, it is not to be wondered at that pigmented spots appear in allergic persons. It is believed that the adrenal cortex controls pigmentation,[18] which accompanies a deficiency of this portion of the gland. Thus in Addison's disease, in which the cortex is destroyed, one finds both lentigines and larger areas of pigmentation.

Both infection and hypoadrenia encourage the formation of lentigo and the two conditions frequently coexist to produce a vicious circle, hypoadrenia favoring infection and infection damaging the adrenal glands further. The increased susceptibility of adrenalectomized (hypoadrenic) animals to infection is well known (Steinach; Pottenger). Children who have inherited or acquired a definite allergy (hypoadrenia) are unfortunate because they are more susceptible to infection.

The most common infection which my associates and I have observed in allergic children is probably chronic sinusitis. This infection is most likely the responsible focus for perennial infectious asthma. The resulting intoxicaton of the adrenal glands may produce many lentigines. This pronounced formation of lentigines, especially in asthma, is believed by Garretson [17] to represent a defense mechanism set up in the skin.

The considerable difference between the numbers of lentigines in comparatively healthy and in ill groups may well represent a corresponding difference between the amounts of cortical hormone from the adrenal glands, which in turn is dependent largely on the presence or the absence of infection and the intensity of infection. It is known that many acute infections quickly damage the adrenal glands, and it is probable that recurring and chronic infections likewise produce hypoadrenia, as evidenced by removal of fat from the adrenal cortex.[19] Comparing the various groups, one finds chronic infections not only in the asthmatic but also in the rheu-

15. Ramond, L.: Diagnostic Study of a Case of Bronchiectasis with Addisonian Melanoderma, Presse méd. **41**:867 (May 27) 1933.

16. Brown, E. E., and Wasson, V. P.: Incidence of Chronic Sinusitis in Rheumatic Children, Arch. Pediat. **59**:735 (Nov.) 1942.

17. Garretson, W. V. P.: Allergy: A Neuroendocrine Interpretation, M. Rec. **141**:269 (March 20) 1935.

18. Grollman, A.: The Adrenals, Baltimore, Williams & Wilkins Company, 1936, p. 86. Moehlig, R. E.: The Pituitary and the Suprarenal Cortex Glands as Related to Pigment Formations, Ann. Int. Med. **4**:1411 (May) 1931.

19. Menten, M. L., and Smith, M. P.: Pathologic Alterations in Lipoid Content of Adrenals in Infancy and in Childhood, Am. J. Dis. Child. **52**:54 (July) 1936.

matic children and in those in the clinic for children with diseases of the chest. The three infected groups show a high incidence of lentigines. In contrast one notes relatively few lentigines in "comparatively healthy" groups. In the group in the general clinic the lentigines were considerably fewer than in the three groups just mentioned. In the children and adults whose colds and infections were infrequent the number was also considerably lower, and no lentigines were present on the newborn infants. The average number of lentigines (28.7) for allergic children is approximately four times as great as the average number for children with rare colds (7 lentigines) or for adults with rare colds (8 lentigines).

The relation between hypoadrenia, infection and lentigines can be extended to include hives and dermographism, conditions sometimes mentioned among the hypoadrenic states. Hives are common in severe malaria, in which lentigines have frequently been mentioned as a complication. Adrenal insufficiency in malaria has been reported by de Brun, Fraga, Garin, Sorony and Pouget.[20] Urticaria, a hypoadrenic condition, is the most common rash in malaria.[21] Although direct sensitivity to the malarial parasites may more commonly be the cause of the associated hives, the possibility of adrenal damage should be considered in those cases in which numerous lentigines develop.

Perhaps the most important evidence associating both lentigines and the melanosis of Addison's disease with lack of adrenal cortex is the depigmentation of these lesions which takes place following the use of a potent extract of adrenal cortex. Grüneberg [22] quoted several observers (Bernard and Thoyer; Bernhardt; Bernreiter; Cantor and Scott; Charpentier; Thompson and Russel; Morawitz) as noting fading of addisonian pigmentation after the use of various extracts of adrenal cortex. He also mentioned Dr. U. Winkler as causing lentigines to become lighter by this therapy, and further, to show the specificity of the extract, declared that it failed to effect depigmentation in chloasma gravidarum. Thorn and co-workers [23] noted a decrease in pigmentation in the majority of patients with Addison's disease following prolonged treatment with a synthetic adrenal cortical factor. Katz and Mainzer [24] caused "freckle-like spots" to become lighter in a case of Addison's disease by a successful adrenal graft.

Relation to Season.—There is apparently nothing in the literature about the seasonal variation in the incidence of lentigines. My observations, however, show a definite increase in the number of lentigines from fall to spring and a lesser decrease from spring to fall. The group observed averaged about 9 years of age and had frequent infections.

In the winter and spring, infections, more common and intense at this time, damage glandular tissue. Damage to the thyroid gland was postulated as the reason for the lower metabolic rates noted in the rheumatic children at this season.[25] It is possible that the same toxin, probably streptococcic, produced in the sinuses, damages the adrenal glands, thus causing increased pigmentation.

20. Fraga, Garin, Sorony, and Pouget, cited by Perla, E. G., and Marmorston, J.: Natural Resistance and Clinical Medicine, Boston, Little, Brown & Company, 1941, p. 476.

21. Thayer, W. C.: Lectures on the Malarial Fevers, New York, D. Appleton and Company, 1899, p. 108.

22. Grüneberg, T.: Effect of Cortical Suprarenal Extracts on Depigmentation, Klin. Wchnschr. **15**:1060 (July 25) 1936.

23. Thorn, G. W.; Dorrance, S. S., and Day, E.: Addison's Disease: Evaluation of Synthetic Desoxycorticosterone Acetate Therapy in One Hundred and Fifty-Eight Patients, Ann. Int. Med. **16**:1053 (June) 1942.

24. Katz, F., and Mainzer, F.: Successful Grafting of Adrenal Gland in Case of Addison's Disease, Brit. M. J. **1**:617 (April 26) 1941.

25. Brown, E. E., and Wasson, V. P.: Basal Metabolism in Rheumatic Children, to be published.

In the fall a correction of the destruction or of the dysfunction of glandular tissue may have taken place. The improved metabolic rates noted at this time suggest more normal thyroid function, and the decreased lentigo count may represent healed or improved adrenal glands.

The pronounced increase in the number of lentigines noted in the 22 children of the clinic for children with diseases of the chest during the winter and spring of 1940 (an average of 13 per patient) may be ascribed to the unusually severe weather that year. During the first six months of 1940, atmospheric temperatures were minus 366 degrees (sum of daily variations from the mean), in contrast to plus 449 and plus 374 degrees of corresponding periods of 1938 and 1939, respectively. Under those severe chilling conditions, toxins, mainly streptococcic, from the upper respiratory tract were produced in abundance. These toxins probably damaged the adrenal cortex. In rheumatic children during the same period Wasson, Brown and Weintraub [26] reported distinct lowering of hemoglobin levels in contrast to the findings of the preceding two winters, probably due to streptococcus toxin. The destruction of hemoglobin, converted to hematin, the increased fragility of capillaries noted in the spring [27] and hypoadrenia were probably the three factors responsible for the great increase in lentigines noted during this cold season.

Relation to Age.—The increase of lentigines with age and the subsequent decrease in old age may be explained as follows: Infections which continue or which recur tend to damage all glandular tissue. The most common chronic infection in human beings is probably chronic suppurative sinusitis, one of the prices paid for evolution (Cullom). Sewall expressed the belief that the great majority of persons harbor infection in the sinuses.[28] The constant absorption of toxin from this source, especially in the winter and spring, may cause increasing damage to the adrenal glands in time, thus possibly accounting for increasing pigmentation, hypotension and weakness, so common in adults in "storm areas."

At middle life and beyond one notes atrophy of all tissues. This is probably true of lentigines also. At this age atrophy is revealed as a physiologic process in the normal graying of hair, ascribed by Hrdlička [29] to absence or diminution in the amount of melanin in the hair. With regard to lentigines, the smaller ones disappear in the atrophy of age. The larger lentigines, moles, may lose not only pigment but also subcutaneous elements and hang as depigmented pedunculated tabs of skin.

Significance of Pigment Formation.—The problem of pigment formation in lentigo is complicated and not solved. The pigment, melanin, is thought to be formed from hematin, a decomposition product of the hemoglobin of red blood cells. The transport of red blood cells or of hematin through capillaries of increased permeability allows the formation of melanin in the skin.

Three factors seem to be present in diseases attended by melanin pigmentation: (1) destruction of hemoglobin (anemia), (2) hypoadrenia and (3) low capillary resistance. This combination of factors probably occurs in Addison's disease, Still's disease, pernicious anemia, rheumatic fever, rheumatoid arthritis and active chronic sinusitis; patients with these diseases are found to show increase of pigmented areas. The first factor, anemia, is common to all these diseases. The second, hypoadrenia, is known to be present in Addison's disease and is

26. Wasson, V. P.; Brown, E. E., and Weintraub, C.: The Blood Picture in Rheumatic Fever, Am. Heart J. **22**:342 (Sept.) 1941.

27. Brown, E. E., and Wasson, V. P.: Capillary Resistance in Rheumatic Children, J. Pediat. **18**:328 (March) 1941.

28. Sewall, E. C.: Abstract of Discussion, J. A. M. A. **111**:1746 (Nov. 5) 1938.

29. Hrdlička, A.: Correspondence, J. A. M. A. **118**:918 (March 14) 1942.

suggested in many cases of the other diseases by findings of hypotension suggestive of hypoadrenia and the pigmentation which is presumably due to this cause. The third factor, low capillary resistance has been found in Addison's disease (Riehl; Ehrmann, quoted by Meirowsky[29]; Kaposi) and in rheumatic fever (Brown and Wasson[27]) and active chronic sinusitis. There is reason to believe it occurs, also, in the other three diseases.

I suggest that circulating toxins from a focus of infection destroy hemoglobin, lower capillary resistance and damage the adrenal glands, both cortex and medulla. The latter contains epinephrine, the depletion of which is necessary for the formation of pigment, according to Goldzieher,[30] who stated:

> Increased skin pigmentation is indicative of a decreased sympathetic tonus or in other words a result of phaeochrome insufficiency. It seems to me that a definite concentration of adrenalin in the circulation is needed to limit the formation of pigment in the skin.

Since epinephrine increases vessel tone, producing spasm, its absence lowers capillary resistance. I have several times improved capillary resistance in hypoadrenic diseases, hives and asthma, within five minutes by injecting 3 minims (0.18 cc.) of epinephrine hydrochloride for these conditions; this improvement was determined with a modified Dalldorf resistometer.

The presence of sufficient epinephrine maintains normal capillary tone and thus breaks the chain in one factor (lowered capillary resistance) of the three which seem to be the sine qua non of pigment formation.

SUMMARY

Many (thirty) distinctions have been noted between lentigines and freckles. These distinctions should be made because of the greater significance of lentigines.

Almost every person past the second year of life has at least 1 lentigo. Lentigines increase in number with age, reaching a peak in adolescence, when more than 30 lesions appear on an average. They tend to diminish in number in middle life and decrease considerably in old age.

Transformations in some of the larger lesions are common in adult life.

Lentigines are probably associated with hypoadrenia, the result of a toxic effect on the adrenal cortex, which controls pigmentation. Both infections and hypoadrenia precede the onset of lentigines.

A relation to toxicity (infection) is suggested by: (*a*) the appearance of lentigines following acute infections; (*b*) their considerable increase in numbers in children with frequent or prolonged colds; (*c*) their hypertrophy to form moles when infections continue; (*d*) their earlier appearance and greater numbers in chronically infected children, especially those with active chronic suppurative sinusitis and bronchiectasis; (*e*) their seasonal variations—their increase in winter and spring in children with frequent colds and their tendency to decrease in summer, when colds do not develop; (*f*) their fewer numbers in children comparatively free from colds and their lack of increase and lesser seasonal variations in these children; (*g*) their reported increase in infectious diseases like rheumatoid arthritis, diphtheria, typhoid fever, malaria, poliomyelitis and some chronic diseases which have many earmarks of infection (Addison's disease, von Recklinghausen's disease, Still's disease, pernicious anemia and chorea).

The relation of lentigines to a possible associated hypoadrenia is suggested by: (*a*) their large numbers in allergic children, especially in those with asthma; (*b*) their usual appearance early in Addison's disease, which is attended by destruction of the adrenal cortex; (*c*) their response (depigmentation of lentigines) to extract of adrenal cortex.

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30. Goldzieher, M. A.: The Adrenals, New York, The Macmillan Company, 1929, p. 276.

BACTERIOLOGY OF FEET AND INCIDENCE OF DERMATOPHYTOSIS OF FEET OF HIGH SCHOOL STUDENTS

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Much has been said about lesions due to fungous infections of the feet and the bacteriology of the feet, and much of what has been said is conflicting. At one time, for example, it was stated that such lesions ("athlete's foot") were found only in frequenters of gymnasiums and then later that as high as 70 to 80 per cent of the male and 50 to 60 per cent of the female population were infected, a large proportion of whom never went near a gymnasium. Also, it has been said by some that fungous infections of the feet are highly communicable and by others that they are not communicable at all. Such statements, and there are many more, indicate that knowledge of the bacteriology of the feet is inadequate.

It seemed desirable to attempt a survey with a group of persons extending over a period of time to try to determine something about the flora of the feet and the frequency of dermatophytosis of the feet. With this in mind, a small high school in Illinois was selected and its students asked to cooperate in the study.

Of 106 students in the high school 60 were willing to participate in the survey. Of these, 33 were boys and 27 were girls. Every two weeks from January 1 to May 30 cultures were made to determine the types of micro-organisms present. Since the children and their families were known well by one of us—who was one of their teachers and their coach—it was thought desirable to try to ascertain any correlation between vocation and family or personal sanitary standards, the bacteriology of the feet and use of the gymnasium and showers and the presence of fungous infection of the feet.

The procedure followed was to call in the student, have him remove his shoes and stockings and then make the culture. The students never knew on what day cultures were to be made; so there was no special preparation or cleansing before samples were taken. Sterile swabs were passed through the interdigital spaces of each foot and then over the

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bottom of the foot from the arch to the toes. The swabs were then streaked thoroughly and carefully over the surface of dextrose agar plates.

Each student was questioned as to whether or not he had or had ever had a fungous infection of the feet ("athlete's foot"). The doubtful reliability of the student answers has been questioned and is admitted. However, it can be said that the junior author had established an excellent rapport, and it is believed that the answers were honest and in good faith. They could have been inaccurate, however, because of inability to recognize the conditions of the feet which were being sought. This fact is admitted and was recognized. It was thought that if a student could give any indications of past infection of the feet, then a more careful laboratory culture could be made. The statements of the students are included here with due recognition of their questionable validity.

At the periods when the swabs were taken the feet were inspected carefully, and again when anything suspicious was noted more careful laboratory cultures were made. In all this work, any time scaly areas on the skin or vesicles were observed scrapings of the skin were cultured. The suspected area was first cleansed with 70 per cent alcohol and then scraped with a sterile scalpel. The material picked up on the scalpel was placed on the surface of Sabouraud acid maltose agar. Microscopic examinations were not made directly from the cutaneous scrapings. In earlier work on the laboratory diagnosis of dermatophytosis of the feet, we had compared the direct microscopic examination of the scrapings digested in sodium hydroxide with the Sabouraud agar plate culture and the sugar broth hanging drop method of Blumenthal and Snow (1936)[1] and had come to the conclusion that the Sabouraud agar plate culture was the most reliable. All cultures were incubated at room temperature for at least one week. After one week's incubation the plates were examined carefully, sometimes with the microscope, and if any growth was apparent they were kept until the growth was sufficient for good observation. The growth of fungi having the general characteristics of Trichophyton, Epidermophyton, etc., was deemed sufficient evidence of the presence of infection.

The bacterial flora was almost entirely composed of Staphylococcus aureus and Staphylococcus albus. These two species were always present and usually in large numbers. This observation confirms other work on the bacteriology of normal skin (Pillsbury, Livingood and Nichols [1942][2]). Occasionally red pigmented micro-organisms closely resembling Serratia marcescens and lactose-fermenting rods resembling

1. Blumenthal, F. S., and Snow, J. S.: A Rapid Culture Method for the Diagnosis of Tinea Infection, J. A. M. A. **107**:1367-1369 (Oct. 24) 1936.

2. Pillsbury, D. M.; Livingood, C. S., and Nichols, A. C.: Bacterial Flora of Normal Skin, Arch. Dermat. & Syph. **45**:61-80 (Jan.) 1942.

Escherichia coli were isolated. The organisms were never found consistently at any one time or in any 1 person. Once in a great while when the feet were very dirty a Bacillus subtilis type of organism was isolated. There were 8 persons with active lesions of dermatophytosis in the group, and no new infections developed during the period of the

TABLE 1.—*Possible Influence of Vocation of Head of Household on Sanitary Habits and the Presence of Dermatophytosis of the Feet*

Head of Household			Number of Students
Miner (28)	Personal sanitary habits	Good	11
		Average	5
		Bad	12
	Family sanitary habits	Good	7
		Average	9
		Bad	12
	Dermatophytosis	Never	12
		Had	12
		Has	4
Farmer (17)	Personal sanitary habits	Good	7
		Average	7
		Bad	3
	Family sanitary habits	Good	8
		Average	4
		Bad	5
	Dermatophytosis	Never	13
		Had	3
		Has	1
W P A. worker (8)	Personal sanitary habits	Good	2
		Average	1
		Bad	5
	Family sanitary habits	Good	1
		Average	1
		Bad	6
	Dermatophytosis	Never	6
		Had	0
		Has	2
Miscellaneous (7)	Personal sanitary habits	Good	4
		Average	1
		Bad	2
	Family sanitary habits	Good	3
		Average	0
		Bad	4
	Dermatophytosis	Never	3
		Had	3
		Has	1

investigation. Five of the patients were boys and 3 were girls. In addition to these 8, 18 students reported having had lesions in the past. Nine were boys, and 9 were girls. In their cases the fungi were never isolated; so the report should be regarded as questionable. The tables give information concerning vocation and family and personal cleanliness, the use of gymnasuim and showers and the presence of dermatophytosis of the feet.

VOCATION

The 60 students were divided into four groups based on the occupation of the head of the household. Three groups, children of miners,

farmers and W P A workers were more or less definite, and the fourth, called miscellaneous, consisted of children of one grade school principal, three widows, one tavern operator and two unemployed persons (one tuberculous). The miscellaneous group showed too much variation in personal and family sanitation to count. This group could have been subdivided with rather significant results showing an interesting difference. Because there were so few cases this was not thought advisable. A survey of table 1 reveals a noteworthy difference in the personal and family sanitary standards of the four groups. The farmers ranked best, miners second and W P A workers third. However, there was no similar difference in the bacteriology of the feet, as the approximate number and kind of bacteria from the feet varied only slightly. With respect to the presence of dermatophytosis of the feet, there is a correlation corresponding to

TABLE 2.—*Comparison of Family and Personal Sanitary Habits and Their Possible Influence on the Presence of Dermatophytosis of the Feet*

		Family Cleanliness			Dermatophytosis		
		Good	Average	Bad	Never	Had	Has
Personal cleanliness	Good (25)	19	4	2	21	3	1
	Average (14)	2	4	8	6	7	1
	Bad (21)	1	3	17	7	8	6
		22	11	27	34	18	8
		Personal Cleanliness			Dermatophytosis		
		Good	Average	Bad	Never	Had	Has
Family cleanliness	Good (22)	19	2	1	16	5	1
	Average (11)	4	4	3	6	4	1
	Bad (27)	2	8	17	12	9	6
		25	14	21	34	18	8

the personal and family sanitary standards; the farm students had fewer fungous infections, the miners' children next and those of the W P A workers most.

PERSONAL AND FAMILY SANITATION

An examination of table 2 reveals a rather close correlation between personal and family sanitary habits and between these conditions and the presence of dermatophytosis of the feet. One (4 per cent) of the subjects with good personal habits had such infection, compared with 1 (7.14 per cent) of the subjects with average personal habits and 6 (28.56 per cent) of the subjects with bad personal habits. The figures for the groups with good, average and bad family habits were the same by number but because of the variation in total numbers varied slightly in percentage. The importance of good sanitary habits in the prevention of fungous infections of the feet is definitely indicated. On the other hand, the general bacterial flora of the feet did not seem to be influenced appreciably by the sanitary conditions.

ATHLETIC INTERESTS

The students were all divided into three groups depending on their athletic interests. The first group did not take physical education and were not engaged in any athletic activity; the second took physical education courses only, and the third took physical education courses and were active in school athletics. The first group were those not coming in contact with the gymnasium dressing rooms and showers, and the last two groups were those using them. Table 3 gives the results of this survey. There were no significant differences between the groups, which indi-

TABLE 3.—*Influence of Athletic Interests on Presence of Dermatophytosis of the Feet*

Interest	Has		Had		Never		Total
	Females	Males	Females	Males	Females	Males	
None	1	2	2	1	7	3	16
Physical education classes	2	1	7	2	8	6	26
Physical education classes and athletics	..	2	..	6	..	10	18
Total	3	5	9	9	15	19	60

TABLE 4.—*Influence of Family Contacts on Presence of Dermatophytosis of the Feet*

Family	Personal and Family Sanitation	Members of Family *		
		1	2	3
1 (2 brothers)	Good	Never (m)	Has (m)	.....
2 (3 brothers)	Below average	Had (m)	Had (m)	Has (m)
3 (2 sisters)	Below average	Has (f)	Has (f)	.....
4 (1 brother, 1 sister)	Good	Never (m)	Had (f)	.....
5 (1 brother, 1 sister)	Good	Never (f)	Had (m)	.....
6 (2 brothers)	Good	Never (m)	Had (m)	.....
7 (2 sisters)	Average	Never (f)	Had (f)	.....

* m indicates male; f, female.

cates that the gymnasium dressing room and shower probably are not the all-important source of dermatophytosis of the feet.

FAMILY CONTACTS

In this group of students there were 15 with brothers or sisters in school. In 6 instances there were 2 children from one family and in 1 instance 3 children. Table 4 gives information concerning sanitary conditions and the presence or absence of fungous infection of the feet in this group. There were 4 subjects with dermatophytosis in the group. Six admitted having had the infection and 5 claimed never to have had it. In 1 family, of below average cleanliness, both sisters attending high

school were infected. In the families of the other 2 families with dermatophytosis other brothers in the family were not infected. In the remaining 4 families fungous infection of the feet was not present, although it is interesting to note that in each family 1 child had had such an infection and the other child had not. This supports the claim of Sulzberger, Baer and Hecht (1942) [3] that "familial and conjugal exposures played no role in eliciting attacks of ringworm on the feet and groins." As before, the importance of sanitary habits of a person and his family is noteworthy.

Sulzberger, Baer and Hecht stated the belief that "alterations in host susceptibility and loss of local immunity are more to blame than new exposures." It seems logical to believe that bad foot sanitation could easily raise host susceptibility or cause a loss of local immunity and thus become an important factor in the acquisition of dermatophytosis. The more one considers infection, especially in relation to diseases of the skin, the more one is led to believe in the old motto that "cleanliness is next to Godliness."

SUMMARY AND CONCLUSIONS

Although the survey was on a small scale, it gave rather definite indications. Apparently the bacterial flora of the feet is almost entirely staphylococci, of both the aureus and the albus species, and within rather wide limits the numbers present are not greatly influenced by the sanitary habits of the subject. Dermatophytosis of the feet was present in 13 per cent of the students examined, which is a figure lower than that usually given. On the other hand, 60 per cent had it or had had it at some earlier time. There was a close relationship between sanitary habits of the subject and the presence of fungous infection of the feet. The disease is not in any way restricted to frequenters of the gymnasium. Surveys of this nature from other types of communities would eventually give valuable knowledge of the bacteriology of the feet.

3. Sulzberger, M. B.; Baer, R. S., and Hecht, R.: Common Fungous Infections of Feet and Groins, Arch. Dermat. & Syph. **45**:670-675 (April) 1942.

SYMMETRIC LIVIDITY OF THE SOLES

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In 1925 Pernet [1] described under the title "Symmetric Lividity of the Soles of the Feet" a disease involving only the soles and characterized by bilateral thickened, edematous, livid plaques which appeared to contain vesicles. Since then Parkhurst [2] has reported 2 cases and cited 2 others previously unreported. Hitch and Hansen [3] termed this dermatosis symmetric erythema of the soles, described 4 new cases in detail and reported having seen 2 others. Sutton and Sutton [4] briefly described the disease.

Authors who have written of symmetric lividity of the soles have expressed the opinion that the dermatosis is not as rare as the number of reported cases would indicate. This opinion was verified in the number of patients with the dermatosis who were seen over a period of a few months at a large military post. During July, August and September, 1942, 18 patients with symmetric lividity of the soles were seen in the dermatology department of the cantonment hospital at Fort Sill, Okla. Of the 18 patients, 17 were white and 1 was a full-blooded American Indian. The disease was not seen among the Negro troops. Because symmetric lividity of the soles was seen so frequently and was misdiagnosed so often, this report is presented.

Since previous communications (Pernet [1]; Parkhurst [2]; Hitch and Hansen [3]) have described cases in detail, only 1 typical case will be reported herein:

REPORT OF A TYPICAL CASE

Two months previous to his first visit to the dermatology clinic, a white soldier, 25 years of age, noted that his heels perspired a great deal, that the skin over the plantar surfaces of the heels had turned white and that his feet had an offensive odor. Since he had had no similar attack previously, and since there was little discomfort except in the morning when he first put on his shoes, he ignored the symptoms at that time. During the month preceding his first visit he noted that the involved areas increased in size, spreading up the sides of the heels, and that the plantar surfaces of the toes had become red and perspired excessively. This aggravation of the dermatosis led him to seek medical aid. Examination of the feet showed horseshoe-shaped lesions extending around the borders of the heels, leaving an uninvolved area in the center. The lesions extended up the sides and the posterior surfaces of the heels for about 3 cm. They were fairly well demarcated and consisted of thickened, white central areas, which appeared to contain numerous vesicles, although none were actually present. The borders of the lesions were violaceous. The plantar surfaces of the toes which were in contact with the shoes also showed violaceous erythema, as did the anterior portion of the balls of the feet. The entire involved area was wet with perspiration. A typical offensive odor was present.

INVESTIGATION OF FOUR CASES

All patients seen were apparently in good health and were able, but for the involvement of the feet, to carry on the duties of a soldier. Therefore, except blood counts, urinalysis and serologic tests for syphilis, no investigations relative to a systemic cause of the disease were

1. Pernet, G.: Symmetric Lividity of the Soles of the Feet, Brit. J. Dermat. **37**:123, 1925.
2. Parkhurst, H. J.: Symmetric Lividity of the Soles, Arch. Dermat. & Syph. **27**:663 (April) 1933.
3. Hitch, J. M., and Hansen, F.: Symmetric Erythema of the Soles, Arch. Dermat. & Syph. **38**:881 (Dec.) 1938.
4. Sutton, R. L., and Sutton, R. L., Jr.: Diseases of the Skin, ed. 10, St. Louis, C. V. Mosby Company, 1939, p. 1052.

carried out. The blood counts and urinalyses gave normal results, and the Kahn test was negative in all cases. After a foot had been surgically prepared, a 6 mm. biopsy punch was used to remove two pieces of tissue in each of 4 cases. One of the pieces of tissue was thoroughly ground up and inoculated on Sabouraud's agar, blood agar and anaerobic brain-heart infusion broth. The other piece of tissue was sectioned and stained for microscopic examination.

Of the blood agar slants, three remained sterile, while the fourth showed a gram-positive bacillus with terminal spores, which was considered a contaminant. At the end of twenty days none of the anaerobic cultures had shown any growth. After twenty-four days one of the cultures on Sabouraud's agar showed colonies of gram-negative bacilli. The other three showed no growth. At no time were fungi found in scrapings prepared with 10 per cent hydroxide. The trichophytin test was applied to 5 patients, 4 of whom showed no reaction at the end of forty-eight hours. The fifth patient had a 1 plus reaction (a papule 0.5 cm. in diameter at the end of forty-eight hours).

The second piece of tissue was sectioned and stained with hematoxylin-eosin for microscopic examination. The sections presented as their most remarkable features edema and vascular dilatation. The vessels of the pars papillaris and the pars reticularis were greatly dilated; they were surrounded by a few round cells, and moderate edema was present. All layers of the epidermis were edematous, while the stratum corneum was greatly thickened and the cells were swollen. In some areas the cells were separated so that microscopic lacunas were visible between them. A few pyknotic nuclei were present. The granules in the stratum granulosum were large, and the layer itself was from four to ten cells in depth. The stratum spinosum was acanthotic and showed intercellular and intracellular edema, as did the stratum germinativum. The sweat glands appeared to be normal.

TREATMENT

Pernet's [1] patients recovered when treated with a sulfur preparation and calamine lotion. Parkhurst [2] obtained relief for his patients by the use of a 25 per cent solution of aluminum chloride. Jamieson [5] cured his patient by using a 1 per cent solution of formaldehyde and a dusting powder. Hitch and Hansen [3] had success with soaks in cold 2 per cent solution of aluminum acetate for one patient, intravenous injections of a solution of calcium thiosulfate for a second and weekly paintings with a 40 per cent solution of formaldehyde for a third. The fourth patient recovered spontaneously after fourteen days' observation. Relapse was apparently uncommon, as only Parkhurst [2] reported a case in which the patient was retreated. Every patient in the present series was instructed to wash his feet daily and put on clean socks at least once a day and a dry pair of shoes each morning. The last instruction was given because perspiration was so profuse that the shoes often did not dry during the night. These patients were treated unsuccessfully with a 1 per cent solution of alum (soaks), alibour water (aqueous solution of zinc and copper sulfates) diluted 3 drachms to a pint of water (25 cc. per liter) (soaks and packs), and a 25 per cent solution of aluminum chloride (repeated paintings). However, the disease responded rapidly when drying powders were used. The most effective of these consisted of 5 per cent powdered alum, 5 per cent tannic acid and 10 per cent boric acid powder in talcum powder. Daily soaks in a 1: 5,000 solution of potassium permanganate proved valuable in conjunction with the drying powder in shortening the course of the disease. The patients were instructed to continue with the use of the powder after all signs of the disease had disappeared. Only a single patient reported to the clinic with a recurrence, which proved to be mild.

COMMENT

The clinical picture of symmetric lividity of the soles is characteristic. This picture consists of bromhidrosis, hyperhidrosis, well demarcated erythematous plaques with sogginess, thickening and apparent but not actual vesiculation involving both soles. Because of the frequency with which the dermatosis was seen among army personnel and because of the frequency with which it was incorrectly diagnosed, it is again called to the attention of the clinician.

The cause of the condition is still not known. Sutton and Sutton [4] described symmetric lividity of the soles in their section on mycotic diseases, although they

5. Jamieson, R. C., cited by Parkhurst.[2]

did not make any definite statement as to the causation. Pernet [1] and Parkhurst [2] did not give an opinion about the causation. Hansen and Hitch,[3] after studying their cases, were of the opinion that the dermatosis should be classified with the erythema multiforme group. One of their patients had erythema multiforme and symmetric lividity of the soles simultaneously. As far as is known at the present time, local infections do not cause the disease. Hitch and Hansen [3] found no fungi on culture or in scrapings. Nolan [6] was not able to culture organisms from the lesions of 5 patients. In the series presented here, no pathologic organisms were obtained from cultures taken from the lesions of 4 patients, and no fungi were found after examining prepared scrapings. The occurrence of the disease during the hot summer months and its absence during the cold months tend to show the seasonal trend. Long marches may play a part in the causation. Cotton-wool socks, which are standard items of issue for soldiers, may contribute to the onset and the prolongation of the disease by keeping the temperature of the feet elevated, as may also the wearing of heavy shoes, which often have a composition rubber sole.

In many ways symmetric lividity of the soles resembles the localized hyperhidrosis sometimes seen on other parts of the body. However, nowhere else does one see with localized hyperhidrosis such decided cutaneous changes as in the disease under consideration. The odor which is such a characteristic feature of symmetric lividity of the soles is also lacking in hyperhidrosis. It is true that an offensive odor may arise from the axillas, the pubic region or even the feet of people who perspire freely and who bathe at infrequent intervals. However, in such cases the odor may often be controlled by frequent cleansing. Simple cleansing is not sufficient to control the odor of symmetric lividity of the soles, possibly because the thickened, edematous stratum corneum is a constant reservoir for the substance which gives rise to the odor. The differences between localized hyperhidrosis seen elsewhere on the body and symmetric lividity of the soles might be explained by the particular location of the latter. Located on the soles, the lesions are subject to trauma, pressure, dependent circulation and poor ventilation. The physiologic ability of the soles to develop great amounts of stratum corneum must also be considered. Until more is known of symmetric lividity of the soles, it is probably best to consider it as a form of localized hyperhidrosis with peculiarities due to its anatomic location. Since it differs so decidedly from other forms of localized hyperhidrosis, it should continue to be differentiated from them.

SUMMARY

Eighteen patients suffering from symmetric lividity of the soles were seen over a three month period at a large army hospital. Attempts to isolate an etiologic organism by culture were unsuccessful, as were attempts to demonstrate fungi by direct examination of prepared scrapings. Possible contributory factors in the causation of symmetric lividity of the soles and the relation of the disease to other forms of localized hyperhidrosis are discussed.

CONCLUSIONS

1. Symmetric lividity of the soles occurs more often than the number of reported cases would indicate.
2. The clinical picture is characteristic.
3. The microscopic picture is not diagnostic.

6. Nolan, L. E.: Personal communication to the author.

4. No pathologic organisms have been demonstrated in the lesions either by culture or by direct examination of scrapings.

5. The disease is probably a form of localized hyperhidrosis but should be differentiated from other forms because of its characteristic clinical picture.

6. The most satisfactory treatment consists of dusting with drying astringent powders in conjunction with soaking in potassium permanganate solution.

Dr. Paul W. Kabler assisted in this work by doing the bacteriologic studies.

THE HISTAMINE TEST

WITH PARTICULAR REFERENCE TO THE DIAGNOSIS OF LEPROSY

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Among the pharmacodynamic tests used for the study of the physiology of the circulation and peripheral innervation, the histamine test is the most important to the dermatosyphilologist. Its usefulness in the differential diagnosis of certain manifestations of leprosy, syringomyelia, polyneuritis and areas of anesthesia of central origin may be great.

The histamine test, first used by Eppinger and Gutmann in 1913, was studied in relation to the peripheral circulation and its disturbances by Sir Thomas Lewis.[1] This test has also been applied to the diagnosis of peripheral neural involvement by Tinel,[2] Loesser [3] and others and of leprosy by Pierini [4] of Buenos Aires, Rodriguez and Plantilla [5] of Manila and Schujman [6] of Rosario. The test consists, according to Lewis, of intradermal injection of a 1 : 1,000 solution of histamine phosphate, the reaction showing in the form of an immediate purpuric spot at the site of the injection followed by an erythematous areola several centimeters in diameter and finally by the formation of a wheal in the center of the erythematous area. This "triple reaction" of Lewis seems to be due to the direct neurovascular stimulation caused by histamine and probably also, as many physiologists believe, to the liberation of histamine, or "H" substance, in the tissues by way of the axon reflex.

The normal reaction to histamine depends on the integrity of the sensory fibers of the peripheral nerves. As these nerves carry both sensory and motor fibers, the association of sensory and motor dis-

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1. Lewis, T. : The Blood Vessels of the Human Skin and Their Responses, London, Shaw & Sons, Ltd., 1927.

2. Tinel, J.: Le système nerveux végétatif, Paris, Masson & Cie, 1937.

3. Loesser, L. H.: The Cutaneous Histamine Reaction as a Test of Peripheral Nerve Function, J. A. M. A. **110**:2136 (June 25) 1938.

4. Pierini: Personal communication to the authors.

5. Rodriguez, J., and Plantilla, F. C.: The Histamine Test as an Aid in the Diagnosis of Early Leprosy, Leprosy Rev. **3**:18 (Jan.) 1932.

6. Schujman, S.: Un nuevo signo diferencial entre siringomielia y lepra nerviosa, Rev. brasil. de leprol. **7**:405 (Dec.) 1939.

turbances is the rule. When a peripheral nerve degenerates as the result of disease or of trauma, the triple reaction does not occur and only the locally produced histamine wheal is observed. After the section or destruction of a nerve, the histamine test elicits a normal response for some time, usually eight to ten days, until the distal end of the nerve degenerates, this phenomenon being explained by the occurrence of axon reflexes still present in the terminal portion of the affected nerve.

In disease of the spinal cord, even though there may be cutaneous anesthesia, the axon reflexes remain unaffected for an indefinite time, and therefore the reaction to the histamine test is normal. In the differential diagnosis of anesthesias and muscular atrophies, particularly those due to leprosy, the histamine test may be most useful in relation to the same symptoms caused by such conditions as syringomyelia, multiple sclerosis and myelitis transversa. According to Loesser,[3] the test is useful also (1) to determine the presence of degeneration and later of regeneration of the nerves following injuries; (2) to differentiate hysterical from organic anesthesia; (3) to differentiate the sensory anesthesias of malingering from the organic; (4) as additional aid in the localization of lesions of the spinal cord, and (5) in the differentiation of involvement of nerve roots from that of the spinal cord.

THE HISTAMINE TEST IN LEPROSY

Our main interest in the histamine test has been in connection with the diagnosis of leprosy. Needless to say, this test would not be necessary with the types of leprosy easily diagnosed by the recognition of clinical symptoms and by the bacteriologic methods usually employed. Its usefulness is undoubted, however, in the early stages of leprosy, when only a few or perhaps only one erythematous lesion may be present, in cases of leprosy affecting the nerves of the extremities with muscular atrophies and to the exclusion of cutaneous lesions, in cases of nonspecific erythematous or dyschromic manifestations and in many cases of tuberculoid leprosy. In the study of contacts and in children, when the truth about normal or abnormal sensations may be difficult to ascertain, the histamine test may be of real help to the dermatologist in detecting the early stages.

The technic of the test as used by us is as follows: A drop of a 1 : 1,000 solution of histamine phosphate is placed on the affected skin; another drop is placed on the border between the normal skin and the suspected area, and a third drop is placed on the normal skin. Through these drops a prick is made with the end of a hypodermic needle, care being taken not to draw blood. As a rule the mere weight of the syringe and needle accomplishes this without exertion of any pressure. The drops of histamine solution are then wiped off with a wad of cotton.

Between twenty-five and forty-five seconds after the prick, the normal skin shows an erythematous areola which spreads in an irregular pattern, reaching a diameter of from 3 to 5 cm. Between sixty and one hundred seconds after the prick, a wheal begins to form on the center of the erythematous area, exactly at the point where the needle penetrated the corium. Finally, after about three minutes, a hemorrhagic spot appears in the needle prick. The wheal measures from 2 to 5 mm. in diameter. The reaction persists for about twenty minutes, but occasionally the erythema and the wheal are still present after forty-five minutes. This sequence of events differs from that reported by Lewis and also by Loesser. There are individual variations due to the age of the subject, to the temperature of the room and to the local state of the circulation. The test is of no value in Negroes.

The reaction to the test performed on the border between the normal and the affected skin shows the same sequence of events toward the healthy skin, but the erythema stops sharply on the boundary and does not appear on the affected side, although the wheal shows equally on both sides.

On the anesthetic skin the erythema is always absent, although the wheal is present as long and is as large as on the unaffected skin.

No subjective sensations are experienced by the subjects under investigation, either on the normal or on the affected areas.

The test performed by scarification or by the intradermal injection of 0.1 cc. of the histamine phosphate solution may be employed also, and some investigators prefer this method, but in our experience the results are not as definite and clearcut as with the method of the prick through the drop.

Similar tests performed with physiologic solution of sodium chloride and with distilled water cause a faint erythema and a punctate wheal at the site of the prick which disappear quickly. These reactions are not constant and may be attributed to the local liberation of "H" substance under the slight trauma caused by the needle.

In the last three years, one of us (F. R. T.) has tested approximately 300 patients with leprosy in the Rincon Leper Hospital, near Habana, and also in the department of dermatology of the Calixto García Hospital. In all these patients the test proved reliable to delimit areas of anesthesia in all types of disease, but particularly those with maculo-anesthetic lesions of the tuberculoid and nonspecific types.

As controls we used about 50 persons suffering from such diseases as acne vulgaris, pediculosis, asymptomatic syphilis, contact dermatitis, lymphogranuloma venereum and pyoderma and other persons with no symptoms of neural or circulatory involvement. In all these subjects the test elicited normal reactions.

It may be added that the temperature of the room while the test is being made must be above 75 F.; when the temperature is below this, normal reactions in normal persons are slow to appear and the flare is less decided, probably because of contraction of the peripheral capillaries.

THE HISTAMINE TEST IN NEUROLOGIC CONDITIONS

Skin locally anesthetized with a 2 per cent solution of procaine hydrochloride without epinephrine was tested, and the results were normal. Apparently the local anesthetic does not abolish the axon reflexes.

In a case of polyneuritis with muscular atrophies of ten years' duration the tests elicited normal reactions, although greatly delayed. In a case of polyneuritis due to avitaminosis B the test elicited negative results, only a wheal resulting after long observation.

In a case of tabes dorsalis in a 48 year old man with early manifestations the tests with histamine resulted in normal reactions.

In a case of traumatic section of the ulnar nerve of several months' duration the histamine test gave a negative result; that is, no erythema was observed and only the locally produced wheal was present. In this case the degeneration of the distal portion of the severed nerve was complete.

In a case of generalized progressive scleroderma histamine tests were performed on the upper and on the lower extremities. All resulted in normal retarded reactions, the erythema appearing after sixty or more seconds and the wheal after three and a half minutes on the average.

In a case of senile arteritis of the extremities with gangrene of the toes the tests resulted in negative reactions, neither erythema nor wheal being observed after five minutes.

We have not been able to apply this test in a case of syringomyelia, a condition often difficult to differentiate from certain types of leprosy. Schujman [6] performed the test in several cases and concluded that in syringomyelia the reaction occurs normally; he stated that the histamine test is a simple, accurate and rapid method to differentiate leprosy and syringomyelia.

CONCLUSIONS

The histamine test is of diagnostic value in cases of peripheral neuritis after degeneration has set in and serves to differentiate anesthetic areas of skin due to peripheral neuritis from those due to disease of the spinal cord. In this connection the test is of great value in the diagnosis of the anesthetic lesions due to leprous neuritis. A normal reaction to the histamine test excludes the diagnosis of leprosy.

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COMBATING LYMPHOGRANULOMA VENEREUM

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Investigations during the past ten years have shown that lymphogranuloma venereum [1] is widespread among the population of the United States. An increase in its incidence is to be expected because of the war. For this reason and in the interest of an efficient conduct of the war, the fight against this disease is of still greater importance now than it has been before. The following suggestions are submitted as a modest contribution to this fight.

SUGGESTIONS FOR RESEARCH ON SO-CALLED LATENT FORMS OF LYMPHOGRANULOMA VENEREUM

The so-called latent forms play an important role in the spread of lymphogranuloma venereum. Nicolau,[2] for example, a most experienced investigator in this field, stated the belief that at least as many infections originate from such forms as from forms with typical lesions. Whether this is an overstatement or not, the obvious fact is that the latent forms of the disease have not as yet received the full measure of attention they deserve, and further investigations of their epidemiologic, clinical and diagnostic aspects are urgently called for in future research on lymphogranuloma venereum. It would be most important to find a standardized method which would enable the diagnostician to differentiate between the contagious type free from typical lesions and the noncontagious type with which there is similarly a positive reaction to a cutaneous test. Investigations should be made especially on women, in whom the latent forms play a much greater role than in men. If a standardized method should be obtained from experimentation on women, it would be automatically just as useful for men. Later in the paper there will be discussion of plans for research on men who have recovered from uncomplicated buboes of lymphogranuloma venereum.

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1. For various reasons, I prefer the old, established name "lymphogranuloma inguinale." However, in order to conform to the rules of the Standard Nomenclature of Disease (Standard Nomenclature of Disease and Standard Nomenclature of Operations, Chicago, American Medical Association, 1942) I use the term "lymphogranuloma venereum" in this paper.

2. Nicolau, S.: Considérations sur la prophylaxie de la lymphogranulomatose inguinale, Bull. Mens. Office internat. d'hyg. pub. **27**:505 (March) 1935.

Present Status of Knowledge.—The following short survey sketches the present knowledge of the so-called latent forms. Clinically one should not take the expression "latent" in a too literal sense. In addition to cases in which there are no pathologic lesions whatsoever, there are others in which are displayed entirely noncharacteristic changes, such as leukorrhea and cervical erosions, and still others in which the lesions are not characteristic enough, such as tiny ulcers and blisters, to be clinically diagnosed as lymphogranuloma venereum even in the presence of a positive reaction to a cutaneous test. Some of these conditions are close to abortive forms; others might later develop into manifest forms, and still others have been manifest in the beginning and have afterward passed into a latent stage. From the epidemiologic point of view, it may be noted that most of the patients with latent forms in whom contagiousness was reported in the literature were females. Single cases of males I found cited were either incompletely described [3] or erroneously included.[4] Nicolau,[2] as well as Coutts, Opazo and Montenegro,[5] who spoke of a saprophytism of the virus of lymphogranuloma venereum in these latent forms, knew such a saprophytic state also in the buccal cavity. Experimentally thus far little work has been done on such latent forms of proved contagious character. Ravaut,[6] as well as Caminopetros,[7] in a few experiments succeeded in infecting a monkey and some mice with material taken from female genitalia. Coutts,[8] as well as Hashimoto and Koyama,[9] prepared effective lymphogranuloma venereum antigens from such material in single cases.

The following paragraphs are brief abstracts of some cases of clinical or epidemiologic interest reported in the literature and short descriptions of the aforementioned experiments.

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3. Yamasaki, I.: Ein Fall von Esthiomène, Jap. J. Dermat. & Urol. **41**:107, 1937.

4. Nair, V. G., and Pandalai, N. G.: Secondary Lymphogranulomatosis of the Anal Margin, Indian J. Ven. Dis. **2**:25 (March) 1936.

5. Coutts, W. E.; Opazo, L., and Montenegro, M.: Digestive Tract Infection by the Virus of Lymphogranuloma Venereum, Am. J. Digest. Dis. & Nutrition **7**:287 (July) 1940.

6. Ravaut, P.: Pathogénie et traitement des rectites hypertrophiantes et sténosantes, Congrès français de chirurgie, Paris, 1934, p. 216.

7. Caminopetros, J.: Recherches épidémiologiques et expérimentales sur la maladie de Nicolas et Favre. Longue persistance du virus de cette maladie dans l'organisme humain, Bull. Soc. path. exot. **28**:408 (June) 1935.

8. Coutts, W. E.: Importance des relations sexuelles anormales dans la propagation des maladies vénériennes et pour la détermination des divers syndromes d'origine sexuelle, Ann. d. mal. vén. **28**:721 (Oct.) 1933.

9. Hashimoto, T., and Koyama, S.: Beiträge zur Kenntnis von Lymphogranuloma inguinale. Ueber den Virusträger, Jap. J. Dermat. & Urol. **38**:133, 1935.

1. Case of Haim and Mathewson Jr.[10]—The last woman to have sexual intercourse with a man afflicted with buboes of lymphogranuloma venereum showed a positive reaction to a cutaneous test, a tiny superficial ulceration of the vulva and a small tender inguinal lymph node. After a few weeks, these minimal clinical signs disappeared without treatment. The patient was advised to return in a month for further examination. (The authors considered the small superficial ulcer observed in the woman as a primary lesion, and they were probably right. However, such tiny, short-lasting ulcerations on the vulva or anus are not always initial lesions. The same type of lesions may appear *after* development of a bubo and may later relapse and even proceed to an esthiomene. Therefore such ulcerations should be observed for a long time before they are considered clinically cured.)

2. Case of Naumann.[11]—A Haitian prostitute, who had served the male youth of her native town in good health for a long time, visited a French steamer in the harbor and soon afterward infected six of her former customers, in whom buboes of lymphogranuloma venereum developed. She herself showed no characteristic lesions but only purulent cervical leukorrhea, a cervical erosion, multiple small blisters at the introitus vaginae and slightly enlarged inguinal lymph nodes. The epidemic stopped with the departure of the woman. (This woman's condition should be called a noncharacteristic rather than a latent form of the disease. About its later development nothing can be said.)

3. Case of Serefis.[12]—On a small Greek island lymphogranuloma venereum became epidemic. All men afflicted with the disease previously had had sexual intercourse with the same prostitute, who did not display visible signs of lymphogranuloma venereum but gave a positive reaction to a cutaneous test. The epidemic expired when the woman was sent to a hospital.

4. Case of Bezecny and Sagher.[13]—A woman who had been operated on for an inguinal bubo four years previously infected her sexual partner with lymphogranuloma venereum and showed no clinical symptoms herself but a positive reaction to a cutaneous test. (If the inguinal bubo operated on four years prior to the transmission of the infection was of lymphogranuloma venereum origin—which is probable—the long duration of the contagiousness of this condition is remarkable.)

5. Experiment of Ravaut.[6]—In the case of a woman with a lymphogranuloma venereum bubo but free of visible lesions of the vagina, Ravaut, in collaboration with C. Levaditi, proved the infectiousness of the vaginal secretion by its inoculation into a monkey brain following passage in guinea pigs.

6. Experiments of Caminopetros.[7]—Caminopetros examined 4 prostitutes who had been sources of lymphogranuloma venereum infections for many men but who themselves were free of visible lesions. He removed from each of them by

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10. Haim, A., and Mathewson, C., Jr.: Incidence of Lymphogranuloma Inguinale in San Francisco, J. A. M. A. **108**:961 (March 20) 1937.

11. Naumann, H. E.: Der klimatische Bubo und seine Therapie, Arch. f. Schiffs- u. Tropen-Hyg. **35**:181 (March) 1931.

12. Serefis, S.: Welche Behandlungsmethoden haben sich bei der Lymphogranulomatosis inguinalis bewährt? Dermat. Wchnschr. **101**:874 (July 13) 1935.

13. Bezecny, R., and Sagher, F.: Orale Infection mit Lymphogranuloma inguinale, Med. Klin. **31**:270 (March 1) 1935.

curettage, after cleaning with sterilized water, tissue of the collum uteri and of the urethra. He then inoculated the eight specimens separately into the brains of a total of eleven mice. In all of the mice meningoencephalitis developed and their brains yielded effective lymphogranuloma venereum antigens. In nine other mice which were inoculated with material taken from three women who did not have lymphogranuloma venereum meningoencephalitis did not develop.

7. Experiment of Coutts.[8]—Coutts prepared an antigen from the vaginal secretion of a woman whose husband suffered from lymphogranulomatous chancre and bubo and produced with this material specific positive cutaneous reactions of lymphogranuloma venereum.

8. Experiments of Hashimoto and Koyama.[9]—These investigators, with the vaginal secretions of 2 prostitutes, obtained results analogous to those of Coutts aforementioned. One of the prostitutes showed swelling and erosion of the cervix and was the source of infection to a man; both prostitutes gave positive reactions to cutaneous tests.

A Plan for Future Research.—Preliminary steps must be the finding of a number of test subjects. These subjects should be women who are known, by case finding evidences, to have been sources of lymphogranuloma venereum infections to males and who themselves give positive reactions to cutaneous tests but show no physical signs of the disease whatsoever or only noncharacteristic changes. Exact histories should be taken and accurate clinical examinations made, including digital exploration of the genitals and rectum, rectoscopy and, if necessary, roentgenographic study of the rectum and colon. The principal interest is centered on those persons who do not show characteristic manifestations of lymphogranuloma venereum anywhere. However, if such subjects are scarce, those showing, for example, rectal involvement need not be entirely omitted, provided that the genital tract has been found free of typical lesions. It would be preferable not to include women suffering from manifestations of other venereal diseases at the same time, especially gonorrhea. Control examinations should be made on women without lymphogranuloma venereum. If test subjects display symptoms such as leukorrhea and cervical erosions, nonlymphogranulomatous persons with analogous symptoms should be selected as controls. In addition, it would be advantageous if further control examinations could be made on women with positive reactions to cutaneous tests whose noncontagiousness has been proved by following up their contacts. From all test and control subjects, material for study should be taken from the cervix, vagina and urethra and, if no suspicious changes are found, from the rectum. The material from each part should be examined separately. Whether secretions or tissues obtained by curettage are preferable and whether curetting should be preceded by washing with sterile saline solution or not will have to be found out by comparison.

1. The first and most important method which should be tried is the animal experiment, that is, intracerebral inoculation of mice.[14] Since the inoculums are contaminated by bacteria and contain, at the same time, substances of human origin, the decision has to be made as to whether they should be used in their original state or after being cleaned by filtration (?), differential centrifugation or subcutaneous passage in guinea pigs. Rodaniche,[15] who worked with contaminated tissues taken from patients with lymphogranulomatous proctitis, had unsatisfactory results with passages in guinea pigs but had only relatively small losses by bacterial infections after direct intracerebral administration of the same material. The mice which survived showed bacterial sterility of the brain. An average of about six mice were used for each inoculum. In order to determine whether meningoencephalitis found in killed mice that have been so inoculated is of lymphogranulomatous origin, tests of three types are available: (1) the cutaneous test of positive reacting human beings and control subjects with antigens prepared from suspected and control brains, (2) the cross immunity tests with mice and (3) the virus neutralization test with mice. The first test represents the simplest way of identifying the virus; the second and third have the advantage that they can be performed entirely in the laboratory. If this complicated preparatory experimentation results in the development of a standardized method, the procedure might be simplified and a reduction might be made in the number of mice required, for example, by limiting the inoculums of each case to material taken from but one or two organs or by pooling the material from different organs for use as a single inoculum. Moreover, it is preferable to perform complicated tests on animals and to sacrifice a large number of mice than to have the contagiousness of a woman proved by the infection of men.

2. The second method to be tried out is the testing of antigens prepared from secretions or tissues of suspected women on positive reactors and control persons. If characteristic cutaneous reactions are obtained, the test could be repeated with specimens taken after the women have been treated with sulfathiazole or a related substance. In order to determine whether such antigens deriving from contaminated specimens are reliable or not, extended control examinations with material prepared from secretions or tissues of women without lymphogranu-

14. Whether it would be possible and, if so, advantageous to replace the intracerebral inoculation of mice by newer methods, such as the intrapulmonary inoculation of rabbits or guinea pigs, intranasal instillation of the material into mice or introduction of the material into the yolk sac of the chick embryo, I am not able to decide, at least not in advance.

15. Rodaniche, E. C.: A Comparison of Seven Strains of the Virus of Lymphogranuloma Venereum, J. Infect. Dis. **69**:138 (Sept.-Oct.) 1941.

loma venereum should be made. Special attention has to be given to the question whether an equivalent of the urethritis chronica nongonorrhoeica Waelsch of men exists in women and, if so, whether secretions of the afflicted women share the attribute of secretions of men with this disease to act like lymphogranuloma venereum antigens in cutaneous tests.[16]

3. A third method is production of immune serums of high titer by injecting yolk sac culture concentrates (Rake, McKee and Shaffer[17]) into suitable animals, to determine whether specific complement fixation or other immune reactions can be obtained with secretions or tissue extracts of suspected women after appropriate preparation.

4. Microscopic examinations of smears and sections and determinations of the albumin-globulin ratio could be performed also.

Whether these extensive investigations will result in the elaboration of a practical and useful method has to be seen. At any rate, the questions involved are important enough to warrant the effort.

SUGGESTIONS FOR RESEARCH ON MEN WHO HAVE RECOVERED FROM UNCOMPLICATED BUBOES OF LYMPHOGRANULOMA VENEREUM

Questions Involved.—In my first paper on the cutaneous test I reported a case of lymphogranuloma venereum with buboes in which the sensitivity to the cutaneous test persisted after recovery.[18] When the number of such observations increased and the opinion was expressed that this persistence was due to a persistence of the causative agent, at that time unknown, I did not accept this theory. I drew a comparison between the cutaneous test for lymphogranuloma venereum and that for chancroid, on which I had worked previously, myself preparing the cultures and vaccines. I had seen that the sensitivity to this test also persisted after convalescence not only in persons with bubo but frequently, although not regularly, likewise in persons with uncomplicated simple chancroidal ulcers, in whom a persistence of the Ducrey bacillus was not probable.[19] I also made a comparison with the artificially

16. (*a*) Frei, W.; Wiese, J., and Klestadt, F.: Harnröhrensekret und Lymphogranuloma inguinale-Reaktion, Klin. Wchnschr. **11**:2114 (Dec. 17) 1932. (*b*) Frei, W.: On the Skin Test in Lymphogranuloma Inguinale: A Brief Review and a Discussion of Some Possible Causes of Error, J. Invest. Dermat. **1**:367 (Oct.) 1938.

17. Rake, G.; McKee, C. M., and Shaffer, M. F.: Agent of Lymphogranuloma Venereum in the Yolk Sac of the Developing Chick Embryo, Proc. Soc. Exper. Biol. & Med. **43**:332 (Feb.) 1940.

18. Frei, W.: Eine neue Hautreaktion bei "Lymphogranuloma inguinale," Klin. Wchnschr. **4**:2148 (Nov. 5) 1925.

19. Frei, W.: Ulcus molle: Bakteriologie, Pathologie, Anatomie, Experimentelles, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1927, vol. 21, p. 1.

produced allergy to primula extract [20] and to neoarsphenamine,[21] in which a long-lasting specific hypersensitivity of the entire skin could be provoked by a single application of a minimal amount to a small area of skin. However, since then the tendency has grown to relate immunity and long-lasting immune reactions associated with virus diseases to a persistence of the virus in the recovered host, and this tendency has also won followers in regard to lymphogranuloma venereum (Favre and Hellerström,[22] Shaffer, Rake and Grace [23] and others). Moreover, it has been demonstrated that the persistence of reactivity in lymphogranuloma venereum is not limited to sensitivity of the skin but extends also to serologic antibodies. Furthermore, it was shown that the virus persists in the brains of infected animals for a relatively long time after convalescence (Jones, Rake and McKee [24] and others) and that in some cases the sensitivity to the cutaneous test can be made to disappear by intensive treatment with sulfanilamide or one of its derivatives (Stein,[25] Harrop, Rake and Shaffer [26] and Axelrod [27]). My opinion at present is that in lymphogranuloma venereum the question of whether or not the persistence of reactivity of the skin and serum after recovery is due to a persistence of the virus has not yet been answered beyond doubt and therefore must be subjected to further investigations. In the meantime Shaffer, Rake and Grace [23] went still a step further with this theory, writing that "it would seem preferable for the present to regard as potential spreaders of virus those persons with evidence of infection whether obtained from clinical findings, skin tests or serological reactions." Although this attitude would undoubtedly prove to be

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20. Bloch, B.: The Rôle of Idiosyncrasy and Allergy in Dermatology, Arch. Dermat. & Syph. **19**:175 (Feb.) 1929.

21. Frei, W.: Ueber willkürliche Sensibilisierung gegen chemisch definierte Substanzen; Untersuchungen mit Neosalvarsan am Menschen, Klin. Wchnschr. **7**:539 (March 18) 1928.

22. Favre, M., and Hellerström, S.: Epidémiologie, étiologie, prophylaxie de la lymphogranulomatose inguinale, Rev. d'hyg. **61**:401 (June-July) 1939.

23. Shaffer, M. F.; Rake, G., and Grace, A. W.: Yolk Sac Antigen in the Diagnosis and Epidemiology of Lymphogranuloma Venereum, Am. J. Syph., Gonor. & Ven. Dis. **26**:271 (May) 1942.

24. Jones, H. P.; Rake, G., and McKee, C. M.: Chemotherapy of Lymphogranuloma Venereum with Sulfonamide Drugs, Proc. Soc. Exper. Biol. & Med. **48**:318 (Oct.) 1941.

25. Stein, R. O.: Observations on Thirty-Five Cases of Venereal Lymphogranuloma Treated with Sulfanilamide, Am. J. Syph., Gonor. & Ven. Dis. **24**:454 (July) 1940.

26. Harrop, G. A.; Rake, G. W., and Shaffer, M. F.: New Clinical Conception of Lymphogranuloma Venereum, Tr. A. Am. Physicians **56**:101, 1941.

27. Axelrod, S. J.: The Diagnosis of Lymphogranuloma Venereum: An Evaluation of a Virus Antigen (Lygranum) Prepared from Culture on Chick Embryo, Am. J. Syph., Gonor. & Ven. Dis. **26**:474 (July) 1942.

the correct one in a great many cases, especially in women (also Favre and Hellerström [22]), the enforcement of so general a procedure would work severe and unwarranted hardships in others. For example, a man who had a bubo of lymphogranuloma venereum of six or eight weeks' duration in his youth would be condemned to permanent celibacy as a potential spreader of virus, since the reactivity of his skin and serum might last indefinitely. For this reason, investigations should be made, especially in that group of men formerly suffering from uncomplicated lymphogranuloma venereum buboes.

Plans for Future Research.—The question whether in lymphogranuloma venereum of human beings the persistence of reactivity of the skin and serum after recovery is due to a persistence of the virus may be approached by the method of mouse brain inoculations. The procedure would be to extirpate small inguinal lymph nodes of men who have histories of buboes of uncomplicated lymphogranuloma venereum, show characteristic scars in the groins and give positive reactions of the skin for lymphogranuloma venereum and negative reactions for chancroid. The inguinal lymph nodes of such men are frequently a little enlarged, especially at the site of the former infection, and one may assume that if the virus is still harbored in the body there are relatively good chances of finding it here. Men displaying such residua of the disease are occasionally found among hospital patients, and the extirpation of a small inguinal lymph node would do no harm. The identification of the virus in a mouse brain would be a matter of routine (aforementioned). The question whether men who have recovered from uncomplicated lymphogranuloma venereum buboes should be considered as potential spreaders of the virus may be approached by investigating their contacts. There are long lists of such persons available in dermatologic clinics and wards and in the files of private practitioners, covering a period of more than ten years. Many of these men have married during this time, and their wives could be tested and clinically examined for lymphogranuloma venereum. Histories of the men should also be brought up to date, and they should undergo testing and clinical examination. Subjects should be carefully selected, and promiscuous groups of the population should be omitted in order to avoid including women who acquired the disease from sources other than their husbands, either before or after marriage. In this respect, wives of former private patients would, on the average, probably be more reliable than wives of clinic patients. The difficulty of getting the consent of the husband for the examination of his wife would be overcome in some of the cases. By cooperation of a large group of dermatologists, a sufficient number of observations might be collected. However, even a small number of carefully selected women would be more valuable than a great number of unreliable ones.

SOME CONSIDERATIONS ON THE CUTANEOUS TEST

In lymphogranuloma venereum, in contrast with the other venereal diseases, the causative agent cannot be identified by simple microscopic or cultural procedures. This want makes the thorough use of every available diagnostic measure indispensable for accurate clinical diagnosis. As the most important of them, I consider the exact clinical examination by experienced physicians. The cutaneous test, which has its merits but also its limitations and which never has been intended to be the only decisive factor in the clinical diagnosis, does not represent more than another important link in the chain of diagnostic measures.

One of the merits of the test is its specificity. Robinson [28] has suspected that the cutaneous test for lymphogranuloma venereum may give false positive reactions in the presence of other venereal diseases. A great number of thorough investigations have been made on that point, some of the best of them in this country. De Wolf and Van Cleve [29] found the test specific in 75 cases of both uncomplicated and complicated chancroidal infections, in 58 cases of uncomplicated gonorrhea and in 84 cases of early active syphilis. Still more imposing are the figures given by Connor, Levin and Ecker,[30] which include those of De Wolf and Van Cleve and contain altogether 315 cases of syphilis with negative cutaneous reactions. Especially syphilis was suspected by Robinson as causing false positive reactions. The only recognized connection between syphilis and the cutaneous test for lymphogranuloma venereum is that in single cases of mixed infections of the two diseases an anergy to the test exists, as stated by myself and confirmed by others (Bacon [31]). Since a high percentage of the patients of De Wolf and Van Cleve apparently belonged to the Negro race—51 of their patients with chancroid, for example, were Negroes—the investigations of these authors also dispelled Robinson's [28a] suspicion that it might be an attribute of this race to produce false positive reactions.

One of the limitations of the test for lymphogranuloma venereum in its original form is based on the difficulties connected with preparing and conserving human antigens. These difficulties may easily be overcome by following my suggestions, "to arrange centralized services to cooperate

28. (*a*) Robinson, H. M.: Analysis of Intracutaneous Tests on Three Hundred and Nine Patients for Lymphogranuloma Venereum and Chancroid, South. M. J. **33**:144 (Feb.) 1940. (*b*) Robinson, H. M., and Robinson, H. M., Jr.: Comparison of Frei Antigens, Arch. Dermat. & Syph. **45**:112 (Jan.) 1942.

29. De Wolf, H. F., and Van Cleve, J. V.: Lymphogranuloma Inguinale, J. A. M. A. **99**:1065 (Sept. 24) 1932.

30. Connor, W. H.; Levin, E. A., and Ecker, E. E.: Observations on the Frei Test, J. Infect. Dis. **60**:62 (Jan.-Feb.) 1937.

31. Bacon, H. E.: The Specificity of the Frei Test in Lymphopathia Venerea, Am. J. Digest. Dis. & Nutrition **2**:570 (Nov.) 1935.

with the different hospitals and clinics and prepare good human vaccine."[32] Sweden, for example, has used such a service with great success for many years, and the experience of the Cleveland City Hospital speaks in the same sense (Brinkley and Love[33]). As long as such services are not established and preparation and control of human antigens lie in the hands of individual investigators, the use of other than human material seems to be preferable, provided that it is prepared by first class laboratories and possesses the same high grade of specificity as adequate bubo antigen. I have had no personal experience with the new chick embryo antigen (lygranum). I admire the splendid experimental work connected with it,[17] and I note the favorable reports hitherto made about its practical value for cutaneous tests (Sulkin, Fletcher, Huber and Reh[34]; Robinson and Robinson Jr.[28b]; Shaffer, Rake and Grace[23]; Combes, Cañizares and Morris,[35] and Axelrod[27]). The new complement fixation test, in which the same antigen has been used (Shaffer, Rake and Grace,[23]) is also very important. However, here one enters an entirely new field, since previous results in complement fixation obtained with human antigens have been unsatisfactory. I have already reported a few negative results in complement fixation in my first paper on the cutaneous test. Further investigations will show whether, and to what extent, the new complement fixation test with chick embryo antigen might be able to replace the cutaneous test. However, it does represent a simple and useful method for first orientation in single as well as mass examinations, provided that positive reactions are still checked and controlled by cutaneous tests. The new complement fixation test might find a special domain in cases in which there is anergy of the skin and perhaps, according to the report of Shaffer, Rake and Grace, also in the early stage of lymphogranuloma venereum when reactions to the cutaneous test are negative.

The importance of lymphogranuloma venereum as a widespread venereal disease demands diagnostic tests on a large scale. The use of the test would be much facilitated if the necessary antigens could be put at the disposal of physicians either without cost or at a low price.

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32. Frei, W.: Venereal Lymphogranuloma, J. A. M. A. **110**:1653 (May 14) 1938.

33. Brinkley, G. W., and Love, W. R.: Mouse Brain Lymphogranuloma Venereum Antigen: Clinical Experience at Cleveland City Hospital, Arch. Dermat. & Syph. **38**:383 (Sept.) 1938.

34. Sulkin, S. E.; Fletcher, P. F.; Huber, E. T., and Reh, E. P.: The Frei Test for Lymphogranuloma Venereum: Experiences with a New Antigen (Lygranum) Prepared from Infected Yolk Sac of Developing Chick Embryo, J. A. M. A. **116**:2663 (June 14) 1941.

35. Combes, F. C.; Cañizares, O., and Morris, G.: Frei Test Evaluation of Chick Embryo Antigen (Lygranum), Arch. Dermat. & Syph. **46**:264 (Aug.) 1942.

In France, the Institut Pasteur has not charged anything for its monkey brain antigen. In Sweden, the State Bacteriological Laboratory has charged 1 krona, formerly the equivalent of 26 cents (now less), for an amount of human antigen sufficient for 1 patient and 1 or 2 control persons. In order to prevent waste, it may be more desirable to follow Sweden's example than to furnish the antigen free of charge.

LYMPHOGRANULOMA VENEREUM ANORECTALE OF MALES AS A DANGER TO MEN HOUSED TOGETHER

Men afflicted with lymphogranuloma venereum of the rectum are under suspicion of pederasty and therefore a special danger as potential sources of infection to men housed together, as in the armed forces. After discovery of the lymphogranulomatous origin of the inflammatory rectal stricture (*"syphilôme anorectal"* Fournier; elephantiasis anorectalis Jersild) by Alice Koppel and myself,[36] the French gastroenterologists Bensaude and Lambling[37] emphasized the importance of pederasty in the pathogenesis of the disease in males.

At the French Congress of Surgery in 1934, which I had the honor of attending by invitation,[38] "Pathogénie et traitement des rectites proliférantes et sténosantes" was one of the main topics. It was presented by the surgeons Gatellier and Weiss[39] and discussed by the many authorities present. Almost all of them agreed on the predominant role of lymphogranuloma venereum in the causation of the disease. As to the role of passive pederasty in the pathogenesis of the rectal infection in men, Bensaude and Lambling[37] presented an imposing array of 96 male patients, of whom not less than 82 per cent confessed passive homosexual practices. These investigators concluded that in men passive pederasty is an especially frequent, if not constant, factor in the pathogenesis of lymphogranuloma venereum of the rectum. A similar opinion was expressed at the same Congress by Rachet and Cachera,[40] who found 14 pederasts among 17 male patients. Later Caminopetros[7]

36. Frei, W., and Koppel, A.: Ulcus vulvae chronicum elephantiasticum (Esthiomène) und sogenanntes Syphilôme anorectal als Folgeerscheinungen der Lymphogranulomatosis inguinalis, Klin. Wchnschr. **7**:2331 (Dec. 2) 1928.

37. Bensaude, R., and Lambling, A.: Considérations sur l'étiologie, la pathogénie et le traitement des rectites présténosantes et des rétrécissements inflammatoires du rectum, Congrès français de chirurgie, Paris, 1934, p. 182.

38. Frei, W.: La preuve du virus lymphogranulomateux vivant dans les tissus des vulvites et rectites proliférantes et ulcérantes est-elle livrée avec sûreté? Congrès français de chirurgie, Paris, 1934, p. 176.

39. Gatellier, J., and Weiss, A.: Pathogénie et traitement des rectites proliférantes et sténosantes, Congrès français de chirurgie, Paris, 1934, pp. 21 and 324.

40. Rachet, J., and Cachera, R.: Pathogénie et traitement des rectites proliférantes et sténosantes, Congrès français de chirurgie, Paris, 1934, p. 238.

of Greece reported that all of his 19 male patients were pederasts (7 professionals and 12 others), and Rajam [41] of India found 8 (?) among 18 patients.

In this country, opinions concerning the importance of pederasty in the pathogenesis of rectal lymphogranuloma venereum in males, are still divided. According to Kornblith,[42] 12 of his 23 patients confessed pederasty. Among 38 thoroughly studied patients of Grace and Henry,[43] 24 acknowledged having had passive rectal intercourse during the period that the lymphogranuloma infection probably occurred. Furthermore, circumstantial evidence indicated that a number of the 14 men who stated that they had not had rectal intercourse were not telling the truth. Therefore, the authors came to the conclusion that in most cases of anorectal lymphogranuloma venereum in males the disease is acquired by direct inoculation of the virus. On the other hand, in Bloom's [44] series of 19 men, few admitted pederasty and many vehemently denied abnormal sexual relations. Bloom [45] expressed the opinion that the theory of the predominant role of pederasty was not based on entirely convincing deductions. He shared the opinion of Gatellier and Weiss [39] that in the majority of cases of rectal involvement in men the disease is acquired by genital inoculation of the virus, which has migrated by way of the lymphatics toward the perirectum and rectum. Also, Levy, Holder and Bullowa [46] obtained a history of pederasty in only a few of their cases.

My personal opinion corresponds to that of Bensaude and Lambling and of Grace and Henry, although my own case list of such types is rather small. In private practice, where on the average more favorable conditions for confidential talks with patients prevail than in clinics, 6 of my 7 male patients afflicted with lymphogranuloma venereum of the rectum confessed passive homosexuality.

Inoculations of lymphogranuloma venereum virus by passive homosexual acts do not necessarily lead to infections of the rectum. Another of my private patients, who was impotent and a passive homosexual at

41. Rajam, R. V.: A Clinical Study of Climatic Bubo and Allied Conditions, Indian M. Gaz. **69**:546 (Oct.) 1934.

42. Kornblith, B. A.: Lymphogranuloma Venereum: Treatment of Three Hundred Cases, Am. J. M. Sc. **198**:231 (Aug.) 1939.

43. Grace, A. W., and Henry, G. W.: The Mode of Acquisition of Lymphogranuloma Venereum of the Anorectal Type, New York State J. Med. **40**:285 (Feb. 15) 1940.

44. Bloom, D.: Lymphogranuloma Venereum: The Present Status, New York State J. Med. **38**:616 (April 15) 1938.

45. Bloom, D., in discussion on Grace and Henry.[43]

46. Levy, J. G.; Holder, E. C., and Bullowa, J. G. M.: Stricture of the Rectum Due to Lymphogranuloma Venereum: Symptoms and Treatment with Sodium Sulfanilyl Sulfanilate, Am. J. Digest. Dis. **9**:237 (July) 1942.

the same time—a combination frequently encountered—acquired only inguinal buboes, due probably to an inoculation of virus into the cutaneous parts of the anus or its surroundings. Another of the same sexual type, who is now symptomless and in the army, has had an isolated lymphogranuloma venereum of the anus. Besides isolated infections of the rectum, anus and superficial inguinal lymph nodes, combinations of two or three of these localizations also occur in connection with homosexual practices.

The involvement of the rectum need not show the picture of a complete rectal stricture or of a rigid proliferative proctitis with or without perianal abscesses or fistulas. In incipient or definitely incomplete infections the mucous membrane of the rectum may be smooth and show only some inflammation or edema or some erosions or ulcerations and produce a slight discharge. In other incomplete infections there may be only an elephantiasic swelling of one or more of the anal folds or some ulcerations at the anus or anal canal. Even isolated perianal abscesses or fistulas without any other sign of rectal or anal involvement occur in connection with such lymphogranuloma venereum infections, although rarely.

Men suffering from fully developed forms of lymphogranuloma venereum of the rectum and anus will scarcely play a role as sources of infection in the armed forces. They will probably not be accepted for service, and, besides, because of the severity of their local lesions they will not be able to perform passive homosexual acts. The danger arises from the incipient or definitely incomplete forms of the disease. Although these facts have certainly been made known to the medical officers by authoritative sources, I should like to call to their attention once more, and to the attention of other physicians, these inconspicuous symptoms of the disease and the necessity of having patients with such incomplete syndromes tested for lymphogranuloma venereum.

SUMMARY

The importance of the latent forms of lymphogranuloma venereum in the spread of the disease is emphasized. A plan for further research on these forms is presented with the principal aim of devising a biologic method to differentiate between contagious and noncontagious infections.

The question whether in lymphogranuloma venereum persistence of positive reactions to cutaneous and complement fixation tests after recovery is due to a persistence of the virus has not been decided yet and should be investigated. Investigations should also be made to determine whether all positive reactors to the tests should be considered "as potential spreaders of virus." Plans for research on these two questions are presented.

The cutaneous test for lymphogranuloma venereum has definite specificity also in the presence of other venereal diseases, especially syphilis, as has been proved by the results of former extended investigations. It is not an attribute of the Negro to have nonspecific reactions. There would not be any difficulty in obtaining sufficient amounts of effective bubo antigen if centralized services should be established. The numerous recommending reports speak in favor of the new chick embryo antigen (lygranum) as a substitute for bubo antigen. Whether the cutaneous test should be replaced by the new complement fixation test with chick embryo antigen must be decided by further investigations.

Men who are afflicted with lymphogranuloma venereum of the rectum and anus are under suspicion of pederasty and are therefore a special danger as potential sources of infection to men housed together, as in the armed forces. The main danger arises from incipient or definitely incomplete forms of the disease. Therefore, even mild symptoms of the rectum and anus should induce medical officers and other physicians to consider the possibility of lymphogranuloma venereum and to make arrangements for testing.

30 Dongan Place.

INCIDENCE OF DERMATOSES IN TWENTY THOUSAND ARMY INDUCTION EXAMINATIONS

WITH A NOTE ON SYPHILIS WITH NEGATIVE SEROLOGIC REACTIONS

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The incidence of diseases of the skin has been studied and recorded by a number of authors. Pollitzer [1] was one of the early ones. He reported his observations covering the period from 1878 to 1911 with a series of 679,376 cases from the records of the American Dermatological Association. The ten most common cutaneous diseases he found, in their order of frequency, were eczema, acne vulgaris, scabies, impetigo, seborrhea, alopecia, pediculosis, tinea, urticaria, psoriasis and verruca. In another series of 58,387 cases from the American Dermatological Association, the same author [2] in 1916 reported the following ten most common diseases of the skin, in their order of frequency: eczema, acne vulgaris, impetigo, psoriasis, scabies, seborrhea, ecthyma urticaria, tinea and dermatitis venenata.

Crocker,[3] reporting on a series of 10,000 cases, stated that the most common diseases of the skin, in order of frequency, were eczema, impetigo, scabies, psoriasis, acne vulgaris and urticaria. Bulkley [4] reported that in a similar series of 10,000 cases the ten most common diseases of the skin, in order of frequency, were eczema, acne vulgaris, psoriasis, dermatitis venenata, scabies, urticaria, pruritus, tinea, seborrhea and impetigo. Goodman,[5] in his concise, excellent article, reviewed the aforementioned reports and added his own from the New York Skin and Cancer Clinic for three periods, 1913 to 1917, 1918 and 1919 and 1923 to 1927. In the last of these periods, study of a series of 108,283 cases revealed that the ten most common diseases of the skin, in order of frequency, were eczema, acne vulgaris, scabies, seborrhea, dermatitis venenata, urticaria, psoriasis, impetigo, verruca and pruritus. Lane [6] recorded statistics on the most common dermatoses encountered in private practice which are in general agreement with those already given, namely, dermatitis, eczema, impetigo, urticaria, acne vulgaris, herpes, verruca, tinea, pruritus and psoriasis, listed in their order of frequency.

From the Recruiting and Induction Station, United States Army, Tacoma, Wash.

1. Pollitzer, S.: Statistical Report of the American Dermatological Association from 1878-1911, J. Cutan. Dis. **32**:312, 1914.

2. Pollitzer, S.: Statistical Report of the American Dermatological Association for the Year 1916, J. Cutan. Dis. **36**:294, 1918.

3. Crocker, H. R.: Diseases of the Skin, Philadelphia, P. Blakiston's Son & Co., 1903, p. 1401.

4. Bulkley, L. D.: Diseases of the Skin, New York, Paul B. Hoeber, 1898, p. 41.

5. Goodman, H.: Statistics of Ten Most Common Skin Diseases, Based on Analysis of 973,090 Published Cases, Arch. Dermat. & Syph. **20**:186 (Aug.) 1929.

6. Lane, C. G.: The General Practitioner and His Work, A. M. A. Bull. **24**:100 (April) 1929.

Gilman,[7] in a survey of diseases of the skin encountered in a student health service, found that in 1,126 diagnoses the ten most common diseases were, in their order of frequency, tinea, acne vulgaris, seborrhea, verruca, pyoderma, dermatitis venenata, eczema, pityriasis rosea, moles and herpes zoster. Alderson and Reich,[8] in a study similar to Gilman's, but on the basis of 5,354 diagnoses, found that the order of frequency and the percentages of the total of the commonest dermatoses were: tinea, 24.7 per cent; pyoderma, 15.4 per cent; dermatitis venenata, 11.8 per cent; seborrhea, 4.4 per cent; acne vulgaris, 3.7 per cent; verruca, 3.5 per cent; sebaceous cyst, 2.9 per cent; urticaria, 2.8 per cent; pediculosis pubis, 1.8 per cent, and herpes simplex, 1.6 per cent. Butterworth and Wilson,[9] reporting the incidence of cutaneous diseases in feebleminded persons from 10 to 30 years of age in a state hospital, found the order of frequency for the ten most common dermatoses to be nevus, tinea, pyoderma, striae distensae, dermatitis traumatica, verruca, hypertrichosis, perlèche, molluscum contagiosum and seborrhea. Seventy per cent of their diagnoses were of the ten most common diseases enumerated. A total of sixty-eight diseases were observed.

TABLE 1.—*Summary of the Incidence of Diseases of the Skin in Nearly 1,000,000 Cases (After Goodman [5])*

Disease Order in Total of 973,090 Cases	Crocker [3] 10,000 Cases	Bulkley [4] 10,000 Cases	Pollitzer [1] 679,376 Cases	Pollitzer [2] 58,387 Cases	S. & C. 44,346 Cases	S. & C. 62,698 Cases	S. & C. 108,283 Cases
Eczema	26.3	26.11	22.5	16.0	20.0	24.0	21.0
Acne vulgaris	4.44	10.44	7.9	7.1	13.0	10.4	10.0
Scabies	7.96	2.13	4.9	3.2	4.0	5.0	5.0
Psoriasis	7.18	3.18	2.8	3.6	4.0	4.1	4.0
Seborrhea	....	....	3.9	3.1	4.0	5.1	5.0
Impetigo	9.61	1.12	3.8	4.3	2.0	5.5	3.0
Urticaria	4.40	0.99	2.9	2.9	5.0	5.1	5.0
Dermatitis venenata	....	1.80	1.6	2.5	3.0	3.0	5.0
Tinea	3.18	2.56	3.2	2.8	3.0	2.0	2.0
Alopecia	....	0.96	3.0	2.1	...	...	...
Pediculosis	....	....	3.4	1.9	...	...	...
Pruritus	0.90	....	1.6	1.2	1.0	0.9	0.8
Verruca	....	1.24	1.0	1.6	1.0	1.5	1.0
Ecthyma	....	1.14	...	3.1	...	...	...

As can be seen, there are great variations in all the statistics, depending on age, sex, race, social level, diagnostic abilities of the earlier dermatologists, seasonal variations, locale and many other factors. Goodman [5] presented an excellent chart of the earlier figures, from 1878 to 1927 (table 1).

The six ranking diagnoses in each group of figures average 50 per cent of all diagnoses, although the six diagnoses vary in each report. Eleven diagnoses equal 57 per cent of all diagnoses for nearly 1,000,000 patients in fifty years. Eczema and acne constitute 30 per cent of all diagnoses and are first and second in all reports cited in table 1 except Crocker's, from England. There are great variations in the statistics of Lane,[6] Goodman,[5] Gilman,[7] Alderson and Reich [8] and Butterworth and Wilson,[9] caused by the many different factors already enumerated. Dermatitis, acne vulgaris, tinea and pyoderma are on all five lists, while eczema, urticaria, herpes zoster, herpes simplex, verruca and seborrhea are on three of the five lists.

7. Gilman, R. L.: Incidence of Skin Diseases in a Student Health Service, Am. J. M. Sc. **188**:268, 1934.

8. Alderson, H. E., and Reich, A.: Incidence of Dermatoses in a Student Health Service, Arch. Dermat. & Syph. **36**:57 (July) 1937.

9. Butterworth, T., and Wilson, M. J., Jr.: Incidence of Diseases of the Skin in Feebleminded Persons, Arch. Dermat. & Syph. **38**:203 (Aug.) 1938.

As far as we have been able to determine, no reports have ever been published on the incidence of dermatoses among men of the 18 to 45 age group who were being inducted into the Army by enlistments and by the Selective Service System. The

TABLE 2.—*The Twelve Most Common Diseases of the Skin and the Rejection Rate of Inductees with These Diseases*

Diseases in Order of Frequency	Number of Cases	Number of Men Accepted	Number of Men Rejected
1. Acne vulgaris	248	248	0
2. Psoriasis	55	44	11
3. Pyoderma	35	35	0
4. Tinea	27	27	0
5. Varicose eczema	20	20	0
6. Neurodermatitis disseminata	20	17	3
7. Lipoma	18	18	0
8. Sebaceous cyst	18	18	0
9. Seborrheic dermatitis	17	16	1
10. Naevus flammeus	17	16	1
11. Neurodermatitis, localized	15	15	0
12. Neurofibromatosis	14	12	2

TABLE 3.—*Incidence of Diseases of Skin and Syphilis in Order of Frequency in 20,000 Men*

Diseases in Order of Frequency	No. of Cases	No. of Men Accepted	No. of Men Rejected
1. Acne vulgaris	248	248	0
2. Psoriasis	55	44	11
3. Pyoderma	35	35	0
4. Tinea	27	27	0
5. Varicose eczema	20	20	0
6. Neurodermatitis disseminata	20	17	3
7. Lipoma	18	18	0
8. Sebaceous cyst	18	18	0
9. Seborrheic dermatitis	17	16	1
10. Naevus flammeus	17	16	1
11. Neurodermatitis, localized	15	15	0
12. Neurofibromatosis	14	12	2
13. Rosacea	14	14	0
14. Contact dermatitis	12	12	0
15. Giant pigmented nevus	12	12	0
16. Scabies	11	11	0
17. Pilonidal sinus	11	8	3
18. Vitiligo	10	10	0
19. Ichthyosis	9	9	0
20. Syphilis of central nervous system	9	0	9
21. Papilloepithelioma	8	8	0
22. Callositas of feet	7	7	0
23. Penile ulceration (nonsyphilitic)	7	7	0
24. Pruritus ani	6	6	0
25. Cutis marmorata	6	6	0
26. Balanitis (nongonorrheal)	6	6	0
27. Flea bites	5	5	0
28. Leukoderma	5	5	0
29. Verruca acuminata	5	5	0
30. Severe smallpox scarring	5	5	0
31. Parapsoriasis	5	3	2
32. Erythema multiforme	4	4	0
33. Lupus erythematosus (chronic discoid)	4	4	0
34. Epithelioma of lip	4	0	4
35. Herpes simplex	3	3	0
36. Urticaria	3	3	0
37. Telangiectasia	3	3	0
38. Alopecia areata	3	3	0
39. Hemangioma cavernosum	3	3	0
40. Latent syphilis	3	0	3
41. Innumerable pigmented nevi	3	3	0
42. Verruca vulgaris	3	3	0
43. Xanthelasma	2	2	0
44. Pityriasis rosea	2	2	0
45. Naevus verrucosus	2	2	0
46. Milia	2	2	0
47. Occupational dermatitis	2	2	0
48. Miliaria	1	1	0
49. Albinism	1	1	0
50. Striae distensae	1	1	0
51. Diffuse melanosis	1	1	0
52. Lichen planus	1	1	0
53. Alopecia totalis	1	1	0
54. Draining cutaneous sinus (nonsyphilitic)	1	1	0
55. Frostbite (congelatio)	1	1	0
56. Clavus (severe)	1	1	0
57. Hidradenitis suppurativa	1	1	0
58. Dermatitis medicamentosa (due to sulfathiazole)	1	1	0
59. Granuloma (nonsyphilitic)	1	1	0
60. Squamous cell epithelioma of eyelid	1	0	1
61. Angioneurotic edema	1	1	0
62. Erythema ab igne	1	1	0
63. Congenital ectodermal defect	1	1	0
64. Keratosis follicularis	1	1	0
65. Fibroma durum	1	1	0
66. Acrocyanosis	1	1	0
67. Generalized arsenical dermatitis (fixed)	1	1	0
68. Epithelioma of penis	1	0	1
69. Generalized combined vascular and pigmented nevus	1	0	1
70. Postherpetic pigmentation (zoster)	1	1	0
71. Prurigo	1	1	0
72. Sarcoma	1	0	1
73. Naevus sebaceus	1	1	0
74. Epidermolysis bullosa	1	0	1
75. Keloid	1	1	0
76. Perlèche	1	1	0
77. Severe hyperhidrosis of face	1	1	0

present series of 20,000 men, who were from the Pacific Northwest, form a cross section of the men of this country at this time, and are a representative group of subjects for the tabulation of cutaneous diseases.

The men when examined were stripped of all clothing and could be surveyed from scalp to toes by the examiner. Dermatophytosis of the feet was not included

in this study, but all other forms of tinea were included. Small moles of the pigmented type were also not included, as they were far too numerous. Pigmented moles of greater size than a silver dollar were included under the term "giant pigmented nevus."

A total of seventy-seven cutaneous diseases were diagnosed, and 733 cases of these various entities were found in the 20,000 men examined. It can be seen from table 2 that six of the most common diseases of the skin in our series are those most frequently noted by other authors. These six diseases are acne vulgaris, psoriasis, pyoderma, neurodermatitis (eczema), tinea and seborrheic dermatitis. Varicose eczema, fifth on our list, is not found in as high a place elsewhere. This may be due to the fact that many of the men were in the age group of 35 to 45, in which this disease is most prevalent. Lipoma, sebaceous cyst and naevus flammeus, in the seventh, eighth and ninth place respectively, are higher on our list than on that of any other author. This may be partly explained by the fact that we reported the diagnoses from our routine observations, while in clinics and private practice few patients come for treatment of such

TABLE 4.—*Incidence of Diseases of the Skin or Syphilis and Percentage of Rejections of Inductees with Such Diseases*

Number of Men Examined	Number of Men with Diseases of Skin or Syphilis	Percentage of Men with Diseases of Skin or Syphilis	Number of Men with Diseases of Skin or Syphilis Fit for Service	Percentage of Men with Diseases of Skin or Syphilis Fit for Service	Number of Men Rejected for Diseases of Skin or Syphilis	Percentage of Total Men with Dermatoses or Syphilis Who Were Rejected
20,000	733	3.66	689	93.99	44	6.01

TABLE 5.—*Rejections for Diseases of the Skin or Syphilis Contrasted with Rejections for Other Causes*

Number of Men Examined	Number Rejected for All Causes	Percentage Rejected for All Causes	Number Rejected for Diseases of Skin or Syphilis	Percentage Rejected for Diseases of Skin or Syphilis
20,000	4,650	23.25	44	0.22

diseases, which consequently are not included in the statistics from these sources. Under acne vulgaris we included 120 cases involving the face, 70 the trunk and 39 the face and trunk. There were 17 cases of acne conglobata and 2 cases of acne indurata. Acne leads our list, as it does all previous ones. Pyoderma in our series included impetigo, ecthyma, furuncles, folliculitis, abscesses and sycosis vulgaris. Under tinea we included 12 cases of tinea versicolor, 10 cases of tinea circinata, 5 cases of tinea cruris and 1 case of erythrasma. The incidence of neurofibromatosis and rosacea, occupying the twelfth and the thirteenth place respectively, is of interest. Contact dermatitis held fourteenth place (tables 2 and 3).

The 9 cases of syphilis of the central nervous system (table 3) detected clinically, including 1 case of dementia paralytica, are of importance, since all these men were selectees and before coming through our station had been reported to have a negative complement fixation or precipitation reaction by their local draft boards. The finding of syphilis of the central nervous system despite a negative serologic reaction of the blood is not unusual with this type of syphilis. Three men with latent syphilis with beginning clinical signs and symptoms of the development of visceral lesions were also rejected, and they, too, had negative serologic reactions for syphilis.

From table 4 it can be seen that 3.67 per cent of the 20,000 men had cutaneous manifestations or syphilis, or 36.7 men out of every thousand examined had a cutaneous disease or syphilis. The incidence of the ten most common diseases of the skin comprises 64.71 per cent of all cases of diseases of the skin found in these 20,000 men. A percentage of 93.99 of men with diseases of the skin were accepted for service, while 6.01 per cent were rejected because of their cutaneous disease or syphilis. From table 2 it can be seen that generalized psoriasis was the greatest cutaneous cause for rejection, with disseminated neurodermatitis second and neurofibromatosis third among the first twelve entities. It is to be noted that the 93.99 per cent accepted for service means the number accepted from a dermatologic standpoint; however, many of them were rejected for other medical reasons. From table 5 it will be observed that the 44 rejections from military service for diseases of the skin or syphilis were insignificant as compared with the 4,650 rejections for all causes, both physical and mental.

SUMMARY

Twenty thousand men from 18 to 45 years of age were examined for military service. Of these, 15,350 were accepted and 4,650 were rejected for all causes. These 20,000 men were found to have seventy-seven cutaneous entities, comprising 733 cases of dermatoses or syphilis. The ten most common diseases of the skin, in order of their frequency (table 2) were acne vulgaris, psoriasis, pyoderma, tinea, varicose eczema, disseminated neurodermatitis, lipoma, sebaceous cyst, seborrheic dermatitis and naevus flammeus. Of all men examined, 3.67 per cent, or 36.7 men per thousand, had a disease of the skin or clinical evidence of syphilis. Of these, 93.99 per cent were fit for service dermatologically speaking, and 6.01 per cent were rejected for their cutaneous disease or for syphilis.

The incidence of the ten most common diseases comprised 64.71 per cent of the total number of cases of dermatoses observed. Generalized psoriasis was the most common cause of rejection, with disseminated neurodermatitis next on the list and neurofibromatosis third. Dermatoses and syphilis together constituted an almost insignificant cause for rejection from military service, there being 44 cases, or 0.22 per cent, among 20,000 men as contrasted with 4,650 rejections, or 23.25 per cent, for all physical and mental causes.

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CHLOROPHYLL IN THE TREATMENT OF ULCERS

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NEW YORK

Chlorophyll, the green coloring material of plants, is not to be found in the animal kingdom. Its chief function, the transformation of light energy into chemical energy, is performed by means of the process known as photosynthesis, the manufacture of carbohydrates from water and carbon dioxide in the presence of light.

The name chlorophyll was given to the green coloring matter of plants by the French chemists Pelletier and Caventou in 1817. Berzelius in 1839 made unsuccessful attempts to determine its chemical structure. Stokes, the British physicist, in 1864 showed that it was a mixture of several chemical substances. Nine years later Sorby separated it into four pigments, two yellow and two green, by partition between immiscible solvents. The work was ignored until Willstätter, who is responsible for most of the knowledge of the chemistry of chlorophyll, repeated and extended the investigation. Between 1906 and 1914 he and his collaborators succeeded in preparing pure chlorophyll for the first time.[1]

In 1930, extracts of plant pigment were shown to have stimulating effects on the growth of tissue by Rollet, working under the direction of E. Bürgi, of Berne, Switzerland, who has conducted a whole series of investigations on this subject during the past twenty years.[2] Gordonoff and Ludwig[3] in 1935 showed that these extracts have a stimulating effect on growth of tissue cultures in vitro. In 1937, in experiments with artificially inflicted wounds on the skin around the spinal column of

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The chlorophyll preparations used in this study were supplied by the Lakeland Foundation.

From the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital. The work was done in collaboration with Dr. J. A. Hyams, of the Department of Urology.

1. West, C. J.: A Review of Willstätter's Researches on Chlorophyll, Biochem. Bull. **3**:229-258 (Jan.) 1914. Steele, C. C.: Recent Progress in Determining the Chemical Structure of Chlorophyll, Chem. Rev. **20**:1-39 (Feb.) 1937. Fischer, H.: Chlorophyll, ibid. **20**:41-68 (Feb.) 1937.

2. Bürgi, E.: Das Chlorophyll als Wachstumsstoff, Klin. Wchnschr. **9**:789 (April 26) 1930.

3. Gordonoff, T., and Ludwig, F.: Ueber den Einfluss der Vitamine auf das Wachstum von Gewebe und von Impfgeschwülsten, Ztschr. f. Vitaminforsch. **4**:213-223 (July) 1935.

rabbits and guinea pigs, Bürgi [4] was able to show that pure chlorophyll has far greater stimulating power over the regeneration of tissue than has either carotene or xanthophyll. In the United States, Gruskin [5] recently reported encouraging results in some 1,200 acute and chronic suppurative conditions treated with chlorophyll.

Because of these considerations, we thought it worth while to attempt to heal chronic ulcers with chlorophyll.

RESULTS OF TREATMENT

The chlorophyll used in this study was prepared in two forms, as an ointment and in aqueous solution. The ointment consisted of oil-soluble derivatives of chlorophyll mixed with hydrous wool fat in the proportion of 1 to 28. The aqueous solution was made by dissolving 2 Gm. of a water-soluble derivative of chlorophyll in a liter of distilled isotonic saline solution.

We selected 25 patients with ulcers of the skin of varied causes who applied for treatment at the Skin and Cancer Unit. The ointment was used topically; the solution was used for wet dressings for ulcers which were surrounded by severe inflammatory zones. There were 10 cases of varicose or hypostatic ulcer, 2 cases of acrodermatitis chronica atrophicans, 1 case of a lesion diagnosed as a roentgen ray ulcer, 6 cases of ulcer attributed to trauma or burn, 1 case of a pyogenic ulcer and 5 cases in which we were unable to find any apparent cause.

Among the 10 varicose or hypostatic ulcers, in 2 cases there was neither granulation tissue nor any other sign of healing, and in 7 there was a rapid filling with granulation tissue. One patient discontinued attendance.

Of the 2 patients whose ulcers were associated with acrodermatitis chronica atrophicans, 1 showed remarkable new granulation tissue; the other discontinued attendance.

The roentgen ray burn healed rapidly in four weeks; it had been treated with various ointments for six months.

All of the 6 ulcers due to trauma or burn showed rapid granulation and healing, but in 1 case a sensitivity to chlorophyll developed and treatment was suspended.

The pyogenic ulcer was the result of an abscess of the buttocks which had broken down and did not heal for six weeks. One week after treatment with chlorophyll was instituted the ulcer shrank to one fourth its original size.

Of the 5 patients with ulcers of unknown origin, 4 showed apparent healing and the fifth discontinued attendance. One of the patients, a

4. Bürgi, E.: Wirkungen von Pflanzenfarbstoffen auf die verletzte Haut, Schweiz. med. Wchnschr. **67**:1173-1176 (Dec. 11) 1937; Ueber die Wirkung von Chlorophyll auf die Wundheilung, ibid. **68**:483-485 (April 30) 1938.

5. Gruskin, B.: Chlorophyll: Its Therapeutic Place in Acute and Suppurative Disease, Am. J. Surg. **49**:49-55 (July) 1940.

woman 60 years of age, had large deep ulcers on both legs. Chlorophyll was used on one leg and the ulcer healed rapidly; on the other leg we used boric acid ointment, and there was no response to the medication.

SUMMARY

Among 25 patients with ulcers of various origin there were 19 who responded favorably to local treatment with chlorophyll used in ointment form or in a 0.2 per cent isotonic aqueous solution. Chlorophyll appeared to have a stimulating effect on the supportive tissues. In some cases the production of granulation tissue in a torpid ulcer was rapid.

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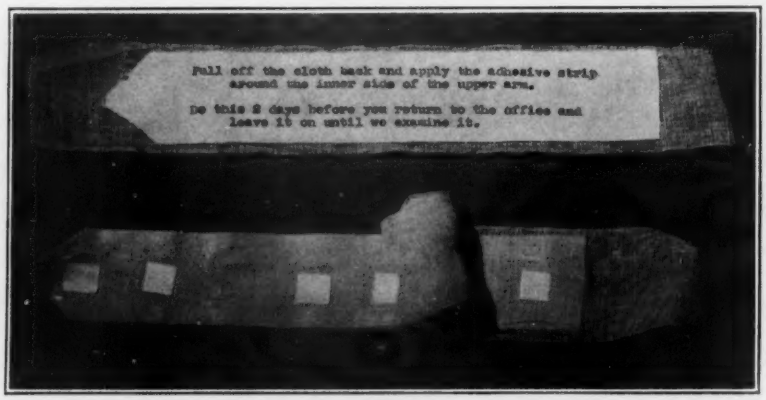
Clinical Notes

PREPARED PATCH FOR CUTANEOUS TESTS

LESTER P. FOWLE, M.D., LEWISBURG, PA.

The prepared patch for cutaneous tests is a time saver and a great convenience. Its chief advantage is that it can be given to a patient with instructions to put it on two or three days before returning to the office. This eliminates the necessity of an extra office visit to read the reaction to the test and saves valuable office time. The patches can be prepared in quantity by a technician or an office assistant. They keep for months. When prepared patches are available one is more likely to give patients cutaneous tests for specific sensitivity to drugs one is planning to use in treatment. Six agents have been used in my routinely prepared patches, but the method can be adapted to include a wider variety of substances.

In preparing the patch I use strips of adhesive tape 1½ inches (4 cm.) wide and about 7 inches (18 cm.) long. One end of the strip is cut to a point, which enables one to tell quickly the location of the no. 1 patch. The patches are small squares of bibulous paper 1 sq. cm. in size. These squares are soaked in solutions of the various agents to be tested,


Pull off the cloth back and apply the adhesive strip
around the inner side of the upper arm.
Do this 2 days before you return to the office and
leave it on until we examine it.

The prepared patch for cutaneous tests.

allowed to dry and then placed in a row on the adhesive tape in the order chosen. Patch 1 is placed close to the pointed end of the strip, and patch 7, which is a blank, is placed near the square end. These patches are easily stored by using a strip of gauze bandage slightly wider and longer than the adhesive, to prevent the adhesive from sticking to itself or to other objects.

I have used the following agents on routine patch strips: (1) sodium arsenate, 10 per cent aqueous solution, (2) copper sulfate, 1 per cent aqueous solution, (3) ammonium fluoride, 1 per cent aqueous solution, (4) mercury bichloride, 1:1,000 aqueous solution, (5) nickle sulfate, 5 per cent aqueous solution, (6) resorcinol,[1] 1 per cent aqueous solution, and (7) control (square of bibulous paper).

Instructions for the use of the patches may be typed on the back of the adhesive strip.

A positive reaction is indicated by a small square area of vesiculation, which is adequate for diagnosis. I have never seen the large angry reactions which develop at times when large wet patches of gauze are used. A few patients have had brisk reactions from the adhesive mass.

None of my patients has ever reacted to all of the agents at one time; so the control is probably not necessary. I continue to use it, however, because it is frequently convenient to have a blank which the patient can use for testing some suspected agent which he has at home in his medicine cabinet.

From Bucknell University.

1. The resorcinol patch slowly turns a light brown due to oxidation. This reaction is slight, however, and does not interfere with the accuracy of the test.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

ECTODERMAL DYSPLASIA OF THE ANHIDROTIC TYPE. HARRY LOWENBURG JR. and ELMER L. GRIMES, Am. J. Dis. Child. **63**:357 (Feb.) 1942.

A patient with an ectodermal dysplasia of the anhidrotic type is described. The following main features were observed: (1) hypotrichosis, (2) absence of sweat glands with intolerance for heat, (3) delayed dentition with roentgenologic evidence of almost complete edentia, (4) failure of lacrimation and (5) atrophic rhinitis. A study of the case reported reveals increasing evidence of the hereditary nature of the disease.

OSTEODYSTROPHIA FIBROSA COMBINED WITH PRECOCIOUS PUBERTY AND EXOPHTHALMIC GOITER. WILLIAM H. STERNBERG and VERA JOSEPH, Am. J. Dis. Child. **63**:748 (April) 1942.

This pathologic report of a case of osteodystrophia fibrosa is of interest to the dermatologist because of the presence of numerous irregularly shaped patches of brown melanotic pigmentation of the skin. When such pigmentation occurs in association with severe, often asymmetric disease of the bone, precocious puberty and occasionally severe exophthalmic goiter the diagnosis of osteodystrophia fibrosa can be made.

TOTAL ALOPECIA ASSOCIATED WITH OCULAR DISORDERS. JOSEPH E. PISETSKY and PHILIP J. KOZINN, Am. J. Dis. Child. **64**:80 (July) 1942.

The authors report a case of total alopecia in a 10 year old white girl associated with pigmentary disturbances of the retina, central scotoma and atrophy of the optic nerve, together with a left internal strabismus. The literature on the association of alopecia areata and various ocular disorders is reviewed.

ORAL PROPHYLAXIS FOR POISON IVY DERMATITIS IN CHILDREN. LEON GOLDMAN, Am. J. Dis. Child. **64**:241 (Aug.) 1942.

This report is concerned with the value of the oleoresin extract of poison ivy as a specific prophylactic agent when given by mouth. After using Shelmire's technic and antigen, the author reports definite progress in producing a specific prophylaxis against poison ivy dermatitis. While no controlled field contact tests were made, only 4 patients out of 39 had poison ivy dermatitis from presumed ordinary exposures. Quantitative patch tests before and after treatment indicated a definite decrease in cutaneous sensitivity.

Cutaneous reactions occurred in a high percentage of cases and included a generalized morbilliform dermatitis, severe pompholyx-like eruption on the palms, extensive papulovesicular dermatitis, a generalized pruritus and pruritus ani.

NELSON PAUL ANDERSON, Los Angeles.

A SYNDROME CHARACTERIZED BY PRIMARY OVARIAN INSUFFICIENCY AND DECREASED STATURE. FULLER ALBRIGHT, PATRICIA H. SMITH and RUSSELL FRASER, Am. J. M. Sc. **204**:625 (Nov.) 1942.

The syndrome discussed by Albright and his associates has little interest for dermatologists, but a large part of the article is devoted to an excellent discussion on hormonal control of axillary and pubic hair. The authors believe that the immediate hormone which stimulates growth of hair in these regions in females comes from the adrenal cortex. They think that ovarian function is not important to the maintenance of axillary and pubic hair.

LYNCH, St. Paul.

THE SPECIFICITY OF PERIARTERIAL FIBROSIS OF THE SPLEEN IN DISSEMINATED LUPUS ERYTHEMATOSUS. IRWIN H. KAISER, Bull. Johns Hopkins Hosp. **71**:31 (July) 1942.

This paper deals primarily with the presence of periarterial fibrosis of the spleen occurring in disseminated lupus erythematosus. Eighteen cases were studied, together with a large number of controls. Periarterial fibrosis of the splenic arteries was found in 15 out of the 18 cases investigated. While this lesion cannot be considered pathognomonic of disseminated lupus

erythematosus, nevertheless, if discovered post mortem it should at least raise the suspicion of that diagnosis. When found in association with the other familiar stigmas of cutaneous, renal and cardiac lesions, periarterial fibrosis in the spleen provides corroborative evidence.

NELSON PAUL ANDERSON, Los Angeles.

HEREDITARY EPIDERMOLYSIS BULLOSA. HARL D. MANSUR JR., J. A. M. A. **120**:1122 (Dec. 5) 1942.

The family history of a 25 year old selectee having hereditary epidermolysis bullosa revealed that 30 of 72 offspring in five generations had the disease. It was proved to be hereditary, did not follow any mendelian ratio but showed dominant characteristics.

Treatment with 5 per cent sulfathiazole ointment applied to the lesions and also a preparation of histaminase, 2 tablets three times a day and at bedtime, did not help the patient.

HENSCHEL, Denver.

COMPARATIVE IMMUNOLOGIC STUDIES WITH SALIVARY AND EPITHELIAL EXTRACTS OF THE DOG, CAT AND RABBIT. W. C. SPAIN, REGINALD E. GILLSON and MARGARET B. STRAUSS, J. Allergy **13**:563 (Sept.) 1942.

The observation that persons hypersensitive to animals frequently exhibit urticaria, localized erythema, itching and edema on areas of skin previously licked or mouthed by pets prompted the authors to investigate the antigenic interrelationship of epithelial and salivary extracts of different animals.

By means of direct cutaneous tests, neutralization point determinations and Dale reactions on sensitized horns of guinea pig uteri, the authors demonstrated that a common antigen is present in the epithelial and salivary extracts of the dog, cat and rabbit. They suggest the possible presence of an extra antigenic fraction in the salivary extract.

A method for the preparation of the extracts is described.

STUDIES IN FOOD ALLERGY: II. SENSITIZATION TO FRESH FRUITS: CLINICAL AND EXPERIMENTAL OBSERVATIONS. LOUIS TUFT and GEORGE I. BLUMSTEIN, J. Allergy **13**:574 (Sept.) 1942.

The authors studied 4 patients suffering from asthma and hay fever in whom the ingestion of raw fruits produced itching or swelling in the mouth or throat. Canned or stewed fruits failed to produce these symptoms. The fruits included apples, bananas, cantaloups, grapes, honeydew melons, oranges, peaches, pears, pineapples and watermelons. Cutaneous tests even with concentrated stock extracts of these fruits constantly gave negative reactions, but strong positive reactions were obtained by tests with freshly extracted juices.

By means of direct cutaneous tests and passive transfer experiments, the authors demonstrated that fresh fruit extracts contain a labile antigen which undergoes rapid deterioration and is either altered or destroyed by heat during both the canning and stewing processes. Rapid freezing (such as the process used for frozen fruits) seems to preserve the allergenic properties of fresh fruits.

The authors suggest that the lability of the antigen in fresh fruit juice probably accounts for the frequent failure to obtain positive cutaneous reactions in clinically sensitive patients even with concentrated stock extracts.

A simple method for the preparation of fresh fruit juices for cutaneous tests is described.

THE TREATMENT OF RHUS POISONING BY ALCOHOLIC EXTRACTS IN A SMALL GROUP CONTROLLED BY PRELIMINARY PATCH TESTS. J. R. CLARKE JR. and C. M. HANNA, J. Allergy **13**:599 (Sept.) 1942.

The authors administered prophylactic treatment against poison ivy dermatitis to a group of 107 boys who were to spend the greater part of the summer of 1941 in a camp where poison ivy was known to flourish. Each boy received a preliminary patch test with a 1 to 500 or 1 to 100 dilution of an alcoholic extract of the plant; the weaker dilution was used in those with a history of previous dermatitis.

Complete data were obtained on only 82 of the group. In 71 of these (86 per cent) the reaction to the patch test was positive; the remainder reacted negatively.

The prophylactic treatment consisted of five subcutaneous injections of an alcoholic extract administered at weekly intervals in increasing doses, the initial dose being 0.05 cc. of a 1 to 100 dilution and the final dose 0.05 cc. of the concentrated solution.

The results were as follows: Seven boys had a rash as severe or more severe than in previous years; 19 had a trifling eruption, and 56 remained entirely free from dermatitis.

MENDELSOHN, New York.

A Quantitative Study of Passive Anaphylaxis in the Guinea Pig. Elvin A. Kabat and Harold Landow, J. Immunol. **43**:69 (May) 1942.

Anaphylactic shock is almost universally believed to be due to the combination of antigen with antibodies present in the tissues of a susceptible animal. Little is known, however, about the actual amounts of antibody which must be present in the tissues in order to cause symptoms of fatal anaphylactic shock on subsequent contact with antigen. The development of quantitative chemical methods for the estimation of precipitins and agglutinins has made it possible to measure how much antibody (in milligrams) must be injected to sensitize a guinea pig and also to study anaphylaxis as a function of the amounts of antigen and antibody necessary for its production.

Experimental work based on these assumptions showed that intravenous injection of 0.03 mg. of rabbit antibody nitrogen will passively sensitize a guinea pig so that fatal anaphylaxis will result on injection of 1 mg. of antigen in the egg albumin system or 0.1 mg. of specific polysaccharide in the type III pneumococcus system. The anaphylactic response is dependent to a great extent on the amount of antigen used. The best anaphylactic response occurs in the region of large antigen excess. The isolated guinea pig uterus has about the same range of sensitivity as the intact animal, though it need contain amounts of antibody of the order of only 0.01 microgram for contraction to be produced. The relation of these findings to the in vitro methods of detecting antibody is discussed.

Antibodies and the Protein-Reserves. Paul R. Cannon, J. Immunol. **44**:107 (June) 1942.

Cannon presents a general theory in an attempt to elucidate further the mechanism of antibody production. This theory is based on the supposition that antibodies are specifically modified globulins which are formed intracellularly during the synthesis of globulin. If this supposition is correct, it follows that antibody production must be influenced by the same conditions which determine globulin production. Inasmuch as experimental evidence indicates that production of globulin is dependent on the intake of amino acids and is impaired by an inadequate intake of dietary proteins, it also follows that antibody production must similarly depend on protein intake. The relationships of the theory to some of the problems related to fluctuations in resistance to infectious disease, to the capacity to produce antibodies and to the conservation of the antibody matrix are discussed. Experimental evidence has been cited indicating, in a preliminary way, at least partial confirmation of the general hypothesis. Some of the implications with respect to the problems of war and mass starvation and the capacity of starving peoples to combat infectious disease are discussed. Cornbleet, Chicago.

Benign Embolic Pyemid. Paul Balog, J. Invest. Dermat. **5**:107 (June) 1942.

Hitherto undescribed dermatosis, "benign embolic pyemid," is described by the author. It is an acute staphylococcic infection, in which a primary banal suppurative focus is followed by repeated pyogenic metastasis. Staphylococcic emboli initiate the lesions by producing localized necrosis. The nodules are characteristically semiglobular and later have an umbilicated necrotic center. The eruption appears suddenly in waves and heals within one to two weeks. The favorite location is the forehead, but it may appear on any part of the upper portion of the body. The absolute benignity of this embolic pyemid distinguishes it from all other septicemic diseases involving the skin.

Studies of the Transmissibility of Syphilis: The Role of the Syphilitic Woman as a Passive Carrier. Harry Pariser, J. Invest. Dermat. **5**:243 (Oct.) 1942.

The author concludes from his experiments, which were interrupted before completion, that Spirochaeta pallida introduced into the vagina of a woman with early syphilis survives only a relatively short time. In 1 instance organisms were recognized morphologically in the vagina until four hours after their introduction, and in another they disappeared within one and one-half hours, after which time the infectiousness of the vaginal secretions could not be demonstrated by animal inoculation.

The Problem of Treatment-Resistant Syphilis: Further Studies on a Treatment-Resistant Strain of Spirochaeta Pallida. Herman Beerman and Marie Severac, J. Invest. Dermat. **5**:269 (Oct.) 1942.

The authors demonstrated that Spirochaeta pallida derived from a treatment-refractory patient still retained a certain amount of the characteristic resistance to treatment when the strain was carried through many animal passages. Bluefarb, Chicago.

IODINE ALLERGY. LOUIS PELNER, J. Lab. & Clin. Med. **27**:1151 (June) 1942.

The author reports on 5 patients with iodine hypersensitivity or allergy which reacted in an unusual way to only a minute quantity of iodine. He distinguishes this reaction from the cutaneous eruption due to prolonged intake of iodine or its salts.

He describes a patch test for the detection of this sensitivity. The test is made with a drop of tincture of iodine, and the reaction is read twenty-four hours later. There were redness and swelling over the site in all the hypersensitive patients tested; nonsensitive persons tested this way did not show this reaction. The reaction was found to be invariable in these patients irrespective of the method of intake of iodine or its salts.

Several types of cutaneous eruptions followed the use of iodine or its salts in these patients, appearing in eight to twelve hours. Three patients showed general reaction to the cutaneously applied tincture of iodine: One had a scarlatiniform eruption; another had a history of such an eruption many years before; the third had thrombopenia purpura, typical in all respects except for the absence of splenic enlargement. The doses of iodine these patients had received were so small that qualitative tests for iodine and iodides gave negative results.

Sodium chloride in 5 to 10 grain (0.3 to 0.65 Gm.) doses seemed to ameliorate the symptoms, the rationale being that the chlorine ion displaces the weaker iodine from the iodide.

The author emphasizes the fact that patients who have iodine allergy must be told of their condition in order not to place their lives in jeopardy at a later date, since there has been an increase in the use of iodides, organic and inorganic, in modern diagnostic and therapeutic technic.

GELBER, Los Angeles.

THE RECOGNITION OF OCCUPATIONAL DERMATOSES. C. GUY LANE, New England J. Med. **227**:39 (July 9) 1942.

Lane stresses the need of careful history taking and careful examination to reach the often difficult conclusion that a given dermatosis is of occupational origin. Causes and clinical manifestations of occupational dermatoses are discussed.

ACUTE DISSEMINATED LUPUS ERYTHEMATOSUS ASSOCIATED WITH FINGER LESIONS RESEMBLING LUPUS PERNIO. J. G. DOWNING and S. J. MESSINA, New England J. Med. **227**:408 (Sept. 10) 1942.

Downing and Messina report the case of a 15 year old girl with lesions of the fingers suggestive of lupus pernio. The reaction to a Hinton test was positive at first and then doubtful. A bismuth compound was administered. A few months later erythematous patches appeared on the bridge of the nose and herpetic lesions on the nose, mouth and palate. A diagnosis was made of lupus erythematosus disseminatus, and the patient died a few months later.

The case is striking because of the early manifestations in the form of apparently benign lesions of the fingers, followed by a rapid change into a fatal disease.

A COMPARISON OF THE TUBERCULIN PATCH TEST WITH THE MANTOUX TEST. H. J. BROCK and F. T. SCHNATZ, New York State J. Med. **42**:1241 (July 1) 1942.

The Mantoux test (1:10,000 old tuberculin intradermally) and the patch test (1 sq. cm. of filter paper prepared by immersion in undiluted old tuberculin solution by Lederle Laboratories) were used in 389 patients.

The authors conclude that the patch test compares favorably with the Mantoux test in determining previous tuberculous infection in children. It is definitely inferior for adults. This may be due to the different texture of the skin of children and of adults.

LEUKEMOID REACTION TO SULFADIAZINE. W. L. WHITTEMORE and M. H. STICH, New York State J. Med. **42**:1249 (July 1) 1942.

A case is reported of a 34 year old diabetic woman with pneumonia in whom the administration of sulfadiazine was effective but produced a leukemoid type of blood reaction, with white blood cell counts as high as 90,400. Discontinuance of administration of the drug and blood transfusion produced a rapid return to a normal blood count.

SKIN IRRITANTS. E. D. OSBORNE and J. J. HALLETT, New York State J. Med. **42**:1529 (Aug. 15) 1942.

Osborne and Hallett discuss the meaning and the use of the terms "primary skin irritant" (a substance which produces primary cutaneous irritation) and "allergen" (a substance producing the reaction of hypersensitivity). They suggest dropping the term "specific irritant."

A patch test made with a primary irritant not properly diluted results in a valueless primary irritation. The clinical features of primary irritation as contrasted with the reaction of sensitization and the most common skin irritants are listed in two charts.

With new chemical compounds the industrial physician should always determine whether these chemicals are primary irritants and the optimum dilution with which to do patch tests. In a third chart are listed the common skin irritants that frequently induce hypersensitivity.

SENSITIZATION. R. L. BAER, New York State J. Med. **42**:1531 (Aug. 15) 1942.

Baer discusses extensively cutaneous sensitization or allergic eczematous dermatitis in industrial occupations, the "host" factors and the contributory outside factors, the frequency and regularity of exposure producing the so-called "hardening" of the skin, the importance of individual difference in thickness of the horny layer of the exposed parts of the skin, the importance of moisture through wetting or hyperhidrosis and the influence of nutritional factors, trauma (friction, burns, cuts, etc.), pyogenic organisms and fungi. He describes also how to reach the proof of sensitization through accurate history taking, study of recurrences, exacerbations and localization of the dermatitis and properly performed patch tests with standard technic.

RONCHESE, Providence, R. I.

IRRADIATION TREATMENT OF CAVERNOUS HEMANGIOMA WITH SPECIAL REFERENCE TO SO-CALLED CONTACT ROENTGEN IRRADIATION. H. DABNEY KERR, Radiology **39**:383 (Oct.) 1942.

The author reports on two series of unselected patients with angiomas. One series of 96 patients was treated with radium, and a second series of 49 was given Chaoul contact therapy. The results in the two series were approximately the same.

The radium technic consists of giving 5 to 7 milligram hours per square centimeter to the lesion at a distance of 2 mm. Ten milligram plaques were employed with a filter of 0.5 mm. steel to 1 mm. platinum. Treatments were given at intervals of six weeks to four months. The dose varied according to the amount of regression.

The technic with the contact therapy apparatus consists of shielding the lesions closely with lead and applying 120 r per minute at 5 cm. distance or 340 r per minute at 3 cm. distance. The greater dose was used on larger lesions. Each dose consisted of 300 to 500 r. The dose was repeated in six to eight weeks. The number of treatments required varied from one to twelve.

ROENTGEN THERAPY OF HYPERTROPHIC SCARS AND KELOIDS. A. F. HUNTER, Radiology **39**:400 (Oct.) 1942.

A study was made of 931 patients with keloids or hypertrophic scars treated with roentgen rays at the Presbyterian Hospital, New York, from 1922 through 1941. The results on the whole were satisfactory, and failures were rare. Most patients were treated with roentgen rays alone. Some were irradiated preoperatively and postoperatively.

At each sitting 200 r is applied. Medium voltage (130 kilovolts peak) radiations with a 3 mm. aluminum filter are applied to closely shielded lesions. The treatment is repeated every two weeks for four sittings. After one or two months elapses a second series of four treatments is given at intervals of two weeks. Not more than four series are given in one year. Two treatments are given preoperatively if the scar is to be excised. The second treatment is given a few days before the operation. Postoperatively four treatments are given as soon as the stitches are removed, to prevent keloid formation.

CIPOLLARO, New York.

SYPHILIS AND THE BIOTROPIC SKIN-EXANTHEMS OF KNOWN BACTERIAL, HEMATOGEN-ENDOGENOUS ORIGIN: SEBORRHEIC DERMATITIS, POMPHOLYX, PITYRIASIS ROSEA, PSORIASIS VULGARIS. TIBOR BENEDEK, Urol. & Cutan. Rev. **46**:409 (July) 1942.

The author states that four syndromes of cutaneous exanthems, i. e., seborrheic dermatitis, pompholyx, pityriasis rosea and psoriasis vulgaris, definitely represent biotropic reactions according to the conception of Milian. The "cryptogenic infection" is a natural one due to transmission of a permanent endoparasite in all human races, Bacillus endoparasiticus (Benedek), from the maternal into the fetal circulation. These biotropic cutaneous reactions may be elicited by arsenic, bismuth or mercury. Whether the eruptions are of shorter or longer duration depends entirely on the more or less continuous or discontinuous bacteremia of B. endoparasiticus (Benedek) released from hidden foci by the bullet shot of antisyphilitic medication. The bullet shot of drug usually elicits only one type of hematogen—endogenous, biotropic cutaneous exanthem. The exanthems, however, may be elicited in any combination of two, three or all four at the same time in the same patient. These four biotropic exanthems are not true drug eruptions, because a micro-organism, B. endoparasiticus (Benedek), is the causative agent of the lesions. Therefore, the author concludes that because the aforementioned cutaneous diseases are merely cutaneous manifestations of biotropism, in those due to antisyphilitic therapy administration of the drug may be continued without interruption.

General X-Ray Baths in Generalized Dermatoses. James Sommerville, Brit. J. Dermat. **54**:234 (Aug.-Sept.) 1942.

The author used generalized irradiation in the treatment of 1 patient with mycosis fungoides and 4 with generalized exfoliative dermatitis. In the case of mycosis fungoides the body was divided into two large fields, and, with the 200 kilovolt plant, a focal skin distance of 200 cm. and 15 milliamperes, it was proposed to deliver 10 r daily to each area, giving 800 r in all over eight weeks. Treatment was suspended after 670 r had been given to the upper half of the body and 710 r to the lower half, because of sickness and anemia. A month later, when the blood picture had improved, a further 200 r was prescribed, with a daily dosage of 20 r three weeks later. Cure was slow, but for the past three years the patient has remained well. It was found in the other cases that with a focal skin distance of 200 cm. and an intensity of 2 r per minute, the resultant blood picture was an anemia of the pernicious anemia type and an extreme lymphopenia, whereas with a focal skin distance of 90 cm. and an intensity of 16 r per minute, the resultant blood picture was almost that of agranulocytosis. The author believes that the safer course lies somewhere between these two.

Lichen Simplex Chronicus (Neurodermatitis of the Palms and Soles). Mary S. Smith, Brit. J. Dermat. **54**:255 (Oct.) 1942.

The author states the opinion that lichen simplex chronicus of the palms has been frequently overlooked. She reports a series of 25 patients, all women. Only 3 of them were under 40 years of age, and the majority were well over 50. Only those were included in whose cases a diagnosis of lichen simplex chronicus of the hands could be confirmed by evidence of lichen simplex chronicus elsewhere. Almost all of them had the lesions on the palms, but 1 or 2 had them on the back of the hands or on the fingers and 2 on the palms and soles. Most of the patients gave a history of related nervous strain. This was particularly noticeable in patients who relapsed under treatment. In differential diagnosis of lichen simplex chronicus from other palmar and plantar keratoses, three points are stressed: the intensity of the itching, which should precede the onset of the eruption; the essential dryness of the lesions and a slight degree of underlying redness. The disease causing most difficulty in differential diagnosis is keratoderma climactericum. The improvement of this condition under mixed glandular therapy, states the author, lends support to the theory of a functional exhaustion of the endocrine glands.

Acne Vulgaris in Infants. Robert Aitken, Brit. J. Dermat. **54**:272 (Oct.) 1942.

The author reports 5 cases of acne vulgaris in infants. The ages of the infants ranged from 4 to 20 months. Two of the patients were girls.

Bluefarb, Chicago.

Pemphigus Foliaceus in Brazil: Its Relation to Senear-Usher Syndrome and European Pemphigus Foliaceus; Preliminary Observations. Rabelo Jr., Arq. de dermat. e sif. de São Paulo **5**:7, 1941.

The author studied cases of pemphigus foliaceus observed in São Paulo and Bello Horizonte, where this disease is endemic. He divides the course of the disease in two periods. The first one is characterized by an erythematous or bullous eruption which is located in the areas usually affected by lupus erythematosus or seborrheic dermatitis. The duration of this stage may be extended up to two years. With the abortive type (Vieira) of the disease the patient recovers during this period. The second stage of the disease is characterized by the exfoliation, which resembles greatly that of the pemphigus of Cazenave; the sign of Nikolsky is always positive. A severe burning sensation is always present, hence the popular name of *"fogo selvagem"* (wild fire). There are severe alopecia and changes in the nails. There are some endocrine disturbances, manifested by atrophy of the breasts and amenorrhea in women.

Dermatomycosis in Buenos Aires. Pablo Negroni, Rev. argent. de dermatosif. **26**:190, 1942.

Negroni in three charts reviews the dermatophytosis observed from 1935 to 1940 in the dermatologic clinic of the Buenos Aires Medical School. Of 34,442 patients seen, 1,900 had dermatophytosis (5.5 per cent). In these statistics he excluded lesions of the folds, keratomycosis and lesions of the mucous membrane. He stated that including those the proportion would be 10 per cent of all the cases. The most frequent lesions observed in men were intertrigo, dermatophytosis of the feet and pityriasis versicolor and in women onychia, paronychia and intertrigo due to blastomycosis. Onychia and paronychia were almost exclusively found in women.

Cañizares, New York.

Society Transactions

BROOKLYN DERMATOLOGICAL SOCIETY

M. M. Estrin, M.D., *President*

Seymour H. Silvers, M.D., *Recorder*

Oct. 19, 1942

A Case for Diagnosis (Aphtha? Vincent's Disease?). Presented by Dr. Abraham Walzer.

V. S., a 53 year old married woman, is presented from the dermatologic clinic of the Beth Moses Hospital. There was nothing in her past history of relevance. She first came to the ear, nose and throat clinic in April 1942, complaining of a sore throat and a sore mouth. Local mouth washes and gargles were ineffectual, and she was referred to the dermatologic clinic on May 9. At that time she showed red areas and abrasions in the mouth, crusting of the lips and crusting at the nasal openings.

Local mouth washes with various substances, such as sodium perborate, and local treatment with various ointments produced no response. On July 23 she was given thiamine hydrochloride, with no effect. On September 29, October 6 and October 13 she was given intravenous injections of neoarsphenamine, 0.3 Gm. per dose, and decided improvement followed.

Tonight she shows crusting on both lips, especially the lower lip, removal of which readily causes bleeding through superficial abrasions. Around the nasal openings also there are some crusts. The mouth, pharynx and tonsillar regions show erythematous areas and some abrasions.

The Wassermann reaction of the blood was negative. The urine was normal. A blood count showed 4,750,000 red cells and 8,800 white cells, with 48 per cent polymorphonuclear leukocytes, 40 per cent lymphocytes, 6 per cent mononuclear cells and 6 per cent eosinophils. Smears showed no bacteria. Cultures of material from the mouth showed Staphylococcus albus and from the throat gram-positive cocci in chains overgrown with Staph. albus.

DISCUSSION

Dr. J. Schweig, New York: I favor the diagnosis of pemphigus. Erosions and ulcerations of the mouth should be viewed with concern, especially if they are of long standing and do not yield to simple measures. I recall several cases in which pemphigus subsequently developed, after a prolonged period of lingering oral lesions. This patient presents pigmented spots on the trunk also, which may have been produced by ingestion of a drug. A toxic eruption due to drugs should be considered. Further observation will be necessary before a diagnosis can be established.

Dr. Jacob Skeer: If this condition were Vincent's disease it would by this time have either become worse or subsided. It is now six months since the onset. If there have been recurrent attacks in the mouth I should suspect pemphigus.

Dr. H. M. Leavitt (by invitation): This patient recalls one whom I saw in the medical ward of Kings County Hospital, whose only complaint was stomatitis as severe as this patient's. The woman was studied carefully; smears and cultures were made, and on only one occasion was the diagnosis of Vincent's infection made. She was given intravenous and intramuscular injections of vitamins and many local applications, without improvement. After three months, however, she began to show typical bullae of pemphigus, and she died soon thereafter. The patient seen here tonight has not improved. Furthermore, the Vincent's angina may be only a secondary infection. The fact that she improved so dramatically after receiving neoarsphenamine may prove that she did have Vincent's stomatitis at the time the drug was administered, but nothing more. At present, the smears show no Vincent organisms, and the only disease to which one can attribute her condition is, I think, pemphigus.

Dr. Max Lerner: This patient was very sick when she first came to the clinic; she could not open her mouth, eat or swallow, and local treatment did little for her. She improved with the administration of vitamins, but her improvement was remarkable after four injections of neoarsphenamine. She did have ulceration. Since she improved after the administration of neoarsphenamine, in spite of the fact that no organism was found, I still think the entire process is Vincent's disease.

Dr. Abraham Walzer: I should like to go over all the diagnostic possibilities presented in this case. When she was seen originally, in May, the patient could barely open her mouth.

The crusting was pronounced and bled easily. The gums showed pyorrhea. The lesions around the nasal orifices and the mouth appeared to be more like abrasions; they were irregular and not sharply defined. The chief possibilities were pemphigus, Vincent's disease and a drug eruption. A history of ingestion of drugs could not be elicited. Local infection or pemphigus was the final diagnosis. The only factor against the diagnosis of Vincent's infection was the absence of organisms on a direct smear; the local manifestations of the disease were there. The only remaining possibility to be considered is pemphigus.

Mycosis Fungoides in a Patient with Carcinoma of the Colon. Presented by Dr. Jacob Skeer.

I. L., a 58 year old white woman, born in Russia, came to the dispensary of the Jewish Hospital complaining of an eruption on the breasts and back of three months' duration.

The patient had been hospitalized on several occasions since November 1938. The diagnoses were diabetes mellitus and generalized arteriosclerosis. She was seen in the dermatologic department in August 1938, at which time she had dermatophytosis and onychomycosis of the hands and feet, as well as isolated vesicles on the hands and legs which were thought to be insect bites. She was admitted to the hospital for operation in February 1942, complaining of cramps of six weeks' duration and of bloody mucous stools. Surgical intervention showed carcinoma of the sigmoid near the splenic flexure, with intussusception. The resected portion of colon showed adenocarcinoma.

The patient was again seen in the dermatologic department on Sept. 2, 1942. Examination showed numerous oval and round erythematous lesions, of various shades of red. These lesions were slightly infiltrated and had fine scales. The eruption was situated on the under surface of the breast, on the chest, the back and the upper part of the abdomen and in the pubic region. The patient complained of severe pruritus.

While in the hospital the patient received various medications, including acetylsalicylic acid, phenobarbital, morphine, insulin, sodium salicylate, theobromine and codeine.

Examination of the blood showed 74 per cent hemoglobin, 3,400,000 erythrocytes, 9,800 leukocytes, 2 per cent band cells, 65 per cent neutrophils, 26 per cent lymphocytes and 7 per cent monocytes. The histologic report was as follows: In places the surface epithelium was greatly attenuated. Within the papillae the tissue was loose and showed infiltration with numerous plasma cells, small round cells and a few polymorphonucleocytes. This pleomorphic infiltration was also seen within the epidermis in certain spots. The histologic diagnosis was mycosis fungoides.

DISCUSSION

Dr. L. M. Fruchtbaum: From the history I think this case is one of mycosis fungoides. I do not believe there is any connection between the carcinoma of the colon and this disease.

Dr. Jacob Skeer: There is little in the literature associating carcinoma with mycosis fungoides, either directly or coincidentally. Whether mycosis fungoides is infectious or not, it is usually fatal. This patient has, in addition, carcinoma of the colon. It is a moot point whether the cancer had anything to do with the development of the mycosis fungoides, and I am unable to answer the question.

Dr. Abraham Walzer: I do not think there is any connection between the two conditions. Mycosis fungoides is primarily a disease of the skin and begins as such. Adenocarcinoma, on the other hand, is not a cutaneous disease.

Poikiloderma of Civatte. Presented by Dr. L. M. Fruchtbaum.

M. R., a woman aged 39, came to the Long Island College Hospital in July 1942 complaining of an extremely pruritic rash on her neck and chest of five years' duration.

There are many macules, papules, scaly lesions and atrophic areas distributed over the upper part of the chest, under the chin and on the sides of the neck and the sides of the lower jaw. The color of the lesions varies from light brown to dark red.

The lesions on the chest are flat, reticulated and brown. The lesions between the jaw and the lower part of the neck are dark red; some are raised, and some are atrophic. Lesions on the sides of the neck up to the lobules of the ears and on the lower jaw are scaly. Hemorrhagic lesions are present on the lips and in the mouth.

The Wassermann reaction of the blood was negative. A blood count was normal. A chemical examination of the blood showed no abnormalities. A histologic examination was reported as favoring poikiloderma.

DISCUSSION

Dr. L. M. Fruchtbaum: I saw this patient three or four times, and each time she presented a different picture. When I saw her five or six years ago the eruption had just begun to

develop. Last July she had no scaling. One month later the papules had disappeared, and my impression was that of poikiloderma. There is no history of local medication, use of cosmetics or ingestion of drugs. The process is gradually extending, and the patient is afraid that the lesion will spread and mar her appearance.

DR. ABRAHAM WALZER: I do not think this patient has poikiloderma. In that disease there are lesions on the face; this patient has none in that location. I should be inclined to call her condition lichen planus.

DR. NATHAN PENSKY: My feeling is that this patient has neither poikiloderma nor lichen planus but, rather, a superficial type of lupus erythematosus. Although the lesions on the chest show no definite atrophy, they are fairly typical of lupus erythematosus. The involvement of the mouth resembles the lesions in this disease, and the definite history of aggravation of the dermatitis on exposure to sunlight is further confirmation of this diagnosis.

DR. JACOB SCHWEIG: I agree with Dr. Walzer that the clinical picture in this case does not conform with the diagnosis presented, as the distribution of the eruption is limited to the uncovered parts of the neck and involves also the vermilion border of the lips. In my opinion the diagnosis is lupus erythematosus with hemorrhagic elements and pigmentation. Biopsy should be repeated.

DR. L. M. FRUCHTBAUM: Civatte described lesions confined to the neck, not to the face. The Jacobi type is generalized over the body. The question is whether this patient's disease is lichen planus or poikiloderma.

Epithelioma and Radiodermatitis of the Arm. Presented by DR. L. M. FRUCHTBAUM.

R. W., a 66 year old man, was first seen by me on June 20, 1942, with the complaint of a large wound on his left arm. He stated he had been suffering from attacks of eruptions around his left elbow for the past twenty-two years. Twenty years ago he received about three roentgen ray treatments per year. In 1938, while in Brazil, he received four roentgen ray treatments every three to four days, each treatment lasting ten to fifteen minutes. Soon afterward hardening of the skin developed in the treated area. About a year ago a small ulcer developed on the irradiated area, which has grown to its present size.

Examination shows an indurated, hard surface of the upper third of the left forearm and its outer aspect and on it an ulcerated and necrotic lesion with proliferated masses over the outer aspect of the elbow bend. The lesion measures about 4 by 3 inches (10 by 7.6 cm.). The histologic report was epithelioma.

DISCUSSION

DR. L. M. FRUCHTBAUM: I should like to know of the experience of other dermatologists with this type of lesion. Is roentgen therapy recommended?

DR. E. A. GAUVAIN: The patient told me he worked in an electrical laboratory, where he was exposed to roentgen rays. From these exposures dermatitis developed, and subsequently cancer. The cancer was treated by roentgen irradiation. Thus, he first had the radiodermatitis, and in this atrophic area cancer developed, which in turn, was treated by roentgen irradiation and then more intensive radiodermatitis developed. I have had no experience with disease as severe as that in this case, but I believe that, with the tremendous amount of scarring present, amputation of the extremity is necessary. It does not seem to me that sufficiently radical surgical therapy can be accomplished otherwise. I suggest that the reason for the lack of metastasis to the regional lymphatics is the intensive scarring, which prevents drainage to the glands.

BRONX DERMATOLOGICAL SOCIETY

DAVID BLOOM, M.D., *President*

HENRY SILVER, M.D., *Secretary*

Oct. 22, 1942

Leprosy (Neural Type). Presented by DR. ADOLPH ROSTENBERG.

The patient, a full-blooded Chinese woman aged 42, is the mother of a child aged 4 years. She came to the city of New York in 1930 and has not left since. The present cutaneous condition began in 1939, with the appearance of a few lesions on the legs. New lesions appeared while some of the older ones disappeared. There were mild itching and slight scaling.

The patient is emaciated but does not complain of pain or internal ailment. The eruption is most pronounced on the lower extremities, especially around the ankles and soles. The lesions

show a fairly symmetric distribution. The face is deeply pigmented. The lesions are round or oval slightly infiltrated reddish patches about 3 inches (7.5 cm.) in diameter; they spread peripherally, becoming depigmented in the center and hyperpigmented at the borders. Some of the lesions have fused, giving to the skin a bizarre appearance. The ulnar nerves are not enlarged and are not sensitive to pressure. Of late, lesions have appeared in the supraorbital region, affecting the median sides of the eyebrows.

Tests for sensibility of the skin in the depigmented centers of the lesions showed considerably diminished tactile perception. The Wassermann reaction of the blood was negative. Examination of the blood, including a cell count, showed no abnormalities.

DISCUSSION

DR. MAURICE UMANSKY: The fact that the patient has spent many years in New York does not speak against the diagnosis of leprosy. There are many physicians who believe that the incubation period may be over thirty years. It is not a highly contagious disease. I have seen 2 patients in Switzerland who were never segregated. I recall a woman aged 25 with ulcerated tumors. She lived in close quarters with the rest of the family, and, during years of observation, no member of the family became infected.

DR. LOUIS CHARGIN: When a patient is extruding lepra bacilli from the nose or open lesions, he must be hospitalized and eventually sent to a leprosarium. But if bacilli are not found after testing, usually with iodides, the patient is not segregated but is kept under observation. However, the case must be reported.

DR. FRANK E. CROSS: It is stated that patients with leprosy have a positive Wassermann reaction. This patient's Wassermann reaction is negative. Perhaps the reason for the large number of positive reactions in leprosy is that the patients have syphilis in conjunction with leprosy.

DR. HENRY SILVER: The subject of the false positive serologic reactions was recently discussed by Drs. Chargin and Rein (The Kahn Verification Test, ARCH. DERMAT. & SYPH. **44**:1031 [Dec.] 1941). They noted that a high percentage of patients with leprosy gave positive serologic reactions.

DR. CHARLES PINES: The Wassermann reaction of lepers is rarely positive in the early stages of the disease. In advanced stages of the disease, especially of the lepromatous type, there is often a positive reaction. But even then it is possible that the patient may be suffering from syphilis or yaws in addition to leprosy.

DR. ADOLPH ROSTENBERG: During a visit to the leprosarium in Habana, Cuba, I was advised that approximately 90 per cent of the patients there have a positive Wassermann reaction. However, it is not certain as to whether this is due to leprosy alone, because many of the patients also have syphilis. Concerning segregation, it was interesting to see that many patients with leprosy were living with their families in close contact in shacks and that rarely did other members become infected. One woman gave birth to a child in the leprosarium. It was taken away to another part of Cuba and remained well until the age of 12 years, at which time it was brought back to the mother and then leprosy developed. Regarding treatment, every patient is treated with chaulmoogra oil. Some react badly, have high temperatures and die, but those who survive seem to be benefited by the fever.

Eruption Due to Vitamin B Complex Deficiency and Precipitated by Prolonged Bismuth Therapy. Presented by DR. PAUL GROSS.

R. V., a white man aged 55, was first seen at the Hospital for Joint Diseases on Sept. 29, 1942. He presented a generalized eruption which had been present for about three months but had spread rapidly prior to admission. The patient had been a heavy whisky drinker up to two years ago and has lived on a limited diet. He has eaten meat and eggs once a week, vegetables twice a week and no milk or butter, and his diet has consisted chiefly of spaghetti with tomato sauce, beans, Italian bread and coffee. For the past year and a half he has noticed a soreness of the mouth, especially of the tongue.

When first seen the eruption consisted of large erythematous scaly patches with a sharp border. These were distributed symmetrically over the backs of the hands, flexor surfaces of the wrists, extensor surfaces of the forearms and arms and posterior surfaces of the thighs and in the gluteal folds. On the face and on the back of the neck were sharply circumscribed patches, especially on the forehead, eyelids and cheeks, suggesting photosensitivity. All the lesions showed edema, which gave them the appearance of moderately infiltrated patches of mycosis fungoides. The patient complained of considerable itching. There was a smooth atrophy and leukoplakia on the anterior portion of the tongue, and the buccal mucosa was covered with plaques. In addition, there was dark bluish discoloration on the tongue, cheeks and gingiva, typical of a bismuth line.

On account of failing vision, he was examined in December 1941 at the Department of Health. Examination of the eyes showed pale atrophic disks with a hazy margin. Vision was 15/100 for the right eye and 3/200 for the left. The Wassermann reaction of the blood was 4 plus. Examination of the spinal fluid showed a 1 plus reaction for globulin, a negative Wassermann reaction and a colloidal gold reaction of 221100000. From December 1941 to September 1942 the patient had received thirty-eight injections of bismuth subsalicylate, and his eyesight had improved promptly.

The patient has received 150 mg. of nicotinic acid daily for two weeks, which has resulted in slight improvement, and a high potency preparation of the vitamin B complex for the past ten days, which has produced a considerable regression of the lesions and great improvement in the patient's physical condition. The lesions on the backs of the hands have cleared, leaving a pigmented and somewhat atrophic skin. The lesions on the extremities have been clearing rapidly, especially in the center.

DISCUSSION

DR. GIRSCH D. ASTRACHAN: I have seen scaly and erythematous eruptions due to bismuth, but I am somewhat doubtful about the cause of the eruption in the case presented. The lesions in some places are infiltrated and sharply defined. This fact and the configuration suggest the diagnosis of mycosis fungoides. With regard to hepatic disturbance due to bismuth, one must admit that such a possibility exists. I have observed a patient in whom there developed a severe hepatitis after bismuth therapy, although catarrhal jaundice could not be ruled out. I have also observed many cases in which the icterus index rose after bismuth therapy.

DR. FRANK E. CROSS: Eruptions due to bismuth are rare, when one considers the number of injections of bismuth compounds that have been given, not only for syphilis but for other diseases of the skin. One has to be careful before making a diagnosis of bismuth eruption. The history of alcoholism in this case suggests that pseudopellagra may account for the dermatitis. All the patches could be part of a vitamin deficiency.

DR. ARTHUR SAYER: The presenter made an interesting point by mentioning the great improvement following vitamin therapy. This fact would indicate that the eruption is due to an avitaminosis. I do not think that a diagnosis of mycosis fungoides should be entertained on the basis of the evidence presented. Certainly one should withhold such an opinion until histologic studies of the tissue present positive proof. I believe that the bismuth therapy did not produce this nonpellagrous cutaneous eruption, and there is no evidence presented to warrant the incrimination of the bismuth as the cause of this eruption.

DR. PAUL GROSS: I assume that the patient had a chronic deficiency with some hepatic damage due to alcoholism and that further damage to the liver by the prolonged bismuth therapy precipitated an acute deficiency syndrome.

Circumscribed Myxedema. Presented by DR. MAURICE UMANSKY.

O. B., a man aged 53, has been suffering from symptoms of exophthalmic goiter since 1939. The basal metabolic rate was +40 per cent. He was operated on in June 1939. A right hemithyroidectomy and isthmectomy were performed. The pathologic report of the specimen submitted for examination was: hyperplastic thyroid, as seen in exophthalmic goiter, in a colloid phase. In July 1939 a thyroidectomy was performed on the left side. The usual procedure of leaving a part of the thyroid was followed. Soon after the operation the patient noticed that the skin over both tibias was becoming dry, scaly and hard. Six to eight weeks later swelling and edema appeared. The swelling of the lower parts of the legs was not permanent at first; it disappeared on rest in bed.

At present a mild degree of exophthalmos is still noticeable. On the lower portion of the right shin there are tumor-like plaques. These are formed through confluence of lesions and present diffuse uneven elevated areas composed of individual hemispheric globular tumors of different size. The plaques are yellowish brown and translucent. No fluctuation is present. On pressure with the finger the skin does not pit. There is one similar patch on the pretibial aspect of the lower portion of the left leg. Slight itching is present. While a specimen was being removed for biopsy a sticky mucilaginous material exuded. The material could be pressed or curetted from the subcutis.

The basal metabolic rate was —23 per cent two months ago. Thyroid preparations have been administered for the last two months. Under this treatment the basal metabolic rate has risen to —10 per cent and the lesions have decreased to about half of their original size.

Chemical examination of the blood showed 39 mg. of urea nitrogen, 9.3 mg. of uric acid, 95 mg. of sugar, 640 mg. of cholesterol, 385 mg. of esterified cholesterol, 525 mg. of sodium chloride, 3.5 mg. of phosphorus and 10.3 mg. of calcium per hundred cubic centimeters. The total protein content of the blood serum was 7.3 mg. per hundred cubic centimeters, the albumin being 5.5 mg. and the globulin 1.8 mg. The carbon dioxide–combining power was 61.2 volumes per cent. The icterus index was 6.

DISCUSSION

DR. PAUL GROSS: The high value for serum cholesterol in this case is undoubtedly the most interesting feature and may have some significance as far as the mechanism of circumscribed myxedema is concerned. The cholesterol content of over 600 mg. per hundred cubic centimeters of serum seems to be out of proportion to a basal metabolic rate of —5 or —10 per cent, which this patient has been reported to show. This would indicate a profound metabolic disturbance resulting from the sudden drop in thyroid function.

DR. CHARLES LERNER: Can the presenter give a possible explanation of the myxedematous change localizing itself to particular areas of the legs and whether or not circulatory obstruction caused by tight garters may have been a factor in causing that localization?

DR. CHARLES WOLF: There are patients who have a hypercholesteremia without evidence of disease. This man may have a high content of cholesterol in the blood which has no relationship to thyroid disase.

DR. MAURICE UMANSKY: This case differs from other recorded cases in that there is a low basal metabolic rate. Also, the chemical examination of the blood shows cholesterol values corresponding to those found in diffuse myxedema, which is always associated with hypothyroidism. Diffuse myxedema responds well to the administration of thyroid, while conditions associated with hyperthyroidism do not. This patient has responded well so far. The plaques have been reduced to half of their original size. So far as I know, circulatory factors do not play a role in localizing the eruption on the legs.

A Case for Diagnosis (Mycosis Fungoides?). Presented by DR. LEO SPIEGEL.

J. P., a white man aged 81, complained of an intensely pruritic eruption, present for the past ten months.

There is a generalized erythematous, slightly scaly eruption with few areas of intervening normal skin. The lesions are of irregular outline and poor definition and resemble eczema or parapsoriasis. The early lesion appears as a flat papule about 0.5 cm. in diameter which fades on pressure. Within these large erythematous areas, which fade but slightly on pressure, are purpuric streaks of various sizes and configurations, the result of scratching. There is a generalized adenopathy.

The urine showed a trace of albumin. The Wassermann reaction of the blood was negative. The white blood cell count was 14,700, with 36 per cent polymorphonuclear leukocytes, 59 per cent small lymphocytes, 3 per cent eosinophils, 1 per cent basophils and 1 per cent myelocytes. The red blood cell count was 3,900,000. There was 80 per cent hemoglobin.

DISCUSSION

DR. FRANK E. CROSS: The lymphocytosis with a differential count of 59 per cent and purpuric tendency suggest an aleukemic eruption. The skin is unusually smooth, which is not generally found in mycosis fungoides. I suggest further studies of the blood.

DR. GIRSCH D. ASTRACHAN: The character of the cutaneous manifestations and the increase in the percentage of lymphocytes suggest a possibility that the lesions belong to the group of lymphoblastomas.

DR. PAUL GROSS: This patient's condition clinically does not fit into the picture of lymphoblastoma of the skin, since it resembles neither leukemia of the skin proper nor leukemic erythroderma. One would be tempted to make the diagnosis of parakeratosis variegata and leave the final decision to the result of a biopsy and bone marrow puncture.

DR. ADOLPH ROSTENBERG: A striking feature in this case is the red color of the face, resembling polycythemia, and the patches on the skin which seemed infiltrated. I consider the condition as one of the lymphoblastomas, but as to whether it is mycosis fungoides or leukemia further study will decide.

DR. LEO SPIEGEL: This eruption, I believe, belongs in the lymphoblastoma group. It is most probably mycosis fungoides. The skin presents various types of lesions which resemble eczema, parapsoriasis, lichenification, ecchymoses due to scratching and various degrees of erythema with slight infiltrations of the erythematous plaques.

NOTE.—Subsequent histologic examination by Dr. D. Satenstein showed stratified squamous epithelium. The surface was intact but irregular in thickness. Just beneath the epithelium the stroma was replaced by a granulomatous lesion. There was little fibroblastic proliferation in the stroma, which was diffusely infiltrated by large and small round cells. Occasionally a few medium-sized "mast cells" and polymorphonuclear leukocytes were noted. A few larger cells, resembling giant cells, were present. The lesion involved the entire thickness of the derma, which contained a number of normal sweat glands. The histologic picture conforms well with the diagnosis of mycosis fungoides.

Lupus Erythematosus Clearing After Tonsillectomy and Sulfanilamide Treatment. Presented by DR. PAUL GROSS.

C. F., a white girl aged 15 years, was first seen at the Hospital for Joint Diseases in March 1941. She has had an eruption since she was 8 years of age, which followed pneumonia. At that time a generalized rash was present, which lasted for a few months. Recurrences have appeared, especially in the summer, but have been confined to the face. She had measles, whooping cough and bronchopneumonia in childhood, and since the onset of menstruation, at the age of 12 years, she has had repeated attacks of suprapubic pain associated with prostration.

The eruption consisted of ill defined erythematous patches on the bridge of the nose and left cheek with a few telangiectases within the lesions. During February and March 1942 the eruption showed signs of spreading and consisted of erythematous slightly edematous and scaly patches in a butterfly distribution on the face. The right tonsil was found to be large, red and tender and a painful enlarged lymph gland was present in the right cervical region.

At present the eruption consists of a faint erythema on the cheeks, on the bridge of the nose and also above the eyebrows. On the elbows and knees there is a follicular xerosis. There are no lesions on the fingers.

Agglutination with hemolytic streptococci was negative. Repeated examinations of the blood from June 1940 on had shown moderate leukopenia, the white cell counts ranging from 3,300 to 5,800. During an attack of subacute tonsillitis the number of white blood cells rose to 7,800. The number of leukocytes decreased to 4,000 after the tonsillectomy. Urinalysis at times showed traces of albumin. The result of a phenolsulfonphthalein test was normal. There never were any visceral signs except a slight enlargement of the spleen. The reaction to a tuberculin test was negative with dilutions of 1 : 1,000,000 to 1 : 10,000.

Sulfanilamide therapy was instituted a few days before the tonsillectomy and was continued for several days after the operation. There was no flare-up of the condition, and the cutaneous eruption improved considerably. During the summer the eruption was barely noticeable and no exacerbations occurred.

DISCUSSION

DR. PAUL GROSS: Before and after the tonsillectomy the patient had been receiving injections of liver extract, which may also have contributed to the improvement. As a rule, one hesitates to remove a focal infection in the presence of lupus erythematosus, especially if it is associated, as in this case, with leukopenia and albuminuria. In cases of this type the possibility of an acute dissemination has to be kept in mind.

DR. HENRY SILVER: I also wish to stress the fact that in the presence of a superficial type of lupus erythematosus in a child, one should be guarded in the prognosis. At the recent symposium at the New York State Medical Society, various aspects of this disease and its pathology were presented. I think that for the time being one should adhere to Klemperer's interpretation of the disease, namely, that the fundamental pathologic change of subacute lupus erythematosus is a fibrinoid degeneration of the collagenous tissue which is similar in all the affected organs (skin, kidney or heart).

DR. ADOLPH ROSTENBERG: I recall a similar case several years ago in which a 14 year old child under my care had a faint flush on the face resembling lupus erythematosus associated with malaise and fever. This followed a recent tonsillectomy. Soon thereafter there developed a fulminating disseminated lupus erythematosus with renal lesions and abscesses in the tonsillar region. After three months the patient died. This case serves to emphasize the serious prognosis in the young.

DR. FRANK E. CROSS: Why was sulfanilamide given before and after tonsillectomy? This case may show the importance of focal infection in some cutaneous diseases. It is possible that a tonsillectomy may prevent generalized dissemination of lupus erythematosus, as exemplified by the case presented.

DR. PAUL GROSS: Sulfanilamide was used to protect against showers of pathogenic organisms which might follow the removal of infected tonsils. It was under these conditions that a tonsillectomy was suggested.

DR. MAURICE UMANSKY: Why is tonsillectomy contraindicated in such a case?

DR. PAUL GROSS: Since the cause of lupus erythematosus is not known, one cannot say what effect the tonsillectomy may have on the disease.

A Case for Diagnosis (Granuloma Annulare? Erythema Annulare Perstans?). Presented by DR. LEO SPIEGEL.

A. K., a white woman aged 42, complains of an eruption limited to the left thigh. During August 1940 she was seen at the clinic of the Lenox Hill Hospital for a lesion on the right

thigh. It was about the size of a silver dollar and had a raised, red, slightly infiltrated border about 0.5 cm. in width. The diagnosis at that time was sarcoid. She disappeared from observation and was not seen again until about three weeks ago. She again complained of an itching eruption, present for the past two weeks, on the left thigh.

On the left thigh there is a ring-shaped erythematous elevated and indurated lesion the size of a palm. The width of the infiltration is about 0.5 cm. The center of the lesion is slightly scaly and hyperpigmented. A similar smaller lesion the size of a dollar is contiguous with the larger ring. On her second visit to the clinic, two weeks later, three new lesions were seen on the left thigh. These lesions are about 0.5 to 1 cm. in size, infiltrated, erythematous and of ring formation.

The Wassermann reaction of the blood was negative.

DISCUSSION

DR. ARTHUR SAYER: Without a biopsy no definite diagnosis can be made in this case. The clinical characteristics are not sufficiently definite to warrant a diagnosis of granuloma annulare.

DR. CHARLES LERNER: At present the large lesion on the back of the thigh does not present a nodular border such as is usually found in granuloma annulare; the absence of this clinical characteristic, in my opinion, speaks strongly against this diagnosis and in favor of erythema perstans.

DR. PAUL GROSS: The small erythematous plaques are elevated as a result of edema, as seen in the erythema group of diseases, and not due to a chronic infiltration, as found in granuloma annulare. The further development into large annular lesions makes the diagnosis of erythema annulare centrifugum more probable.

DR. DAVID BLOOM: The border of the lesion does not consist of individual nodules but forms a continuous infiltration. This case resembles closely that of a young girl who had a similar lesion on the back of the right thigh for more than a year and who was presented by Dr. Abramowitz before the Manhattan Dermatologic Society a year ago (ARCH. DERMAT. & SYPH. **45**:779 [April] 1942). I consider this case as one of erythema annulare perstans.

CLEVELAND DERMATOLOGICAL SOCIETY

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JAMES R. DRIVER, M.D., *Reporter*

Oct. 22, 1942

Pemphigus Vulgaris. Presented by DR. W. R. HUBLER.

G. B., a Russian Jewess aged 38, presented from the department of dermatology and syphilology, University Hospitals, was admitted to Lakeside Hospital in September 1942 because of bullous lesions scattered over the skin. The onset was heralded by the appearance of a bulla in the umbilicus three months ago. It was not until seven weeks later that she noticed several new blebs appearing on her back and chest. Three weeks before admission she began to have many painful ulcerated areas in her mouth. Recently she has felt considerable general malaise. She has lost 30 pounds (13.6 Kg.) since the onset of the disease.

She has an ulceration in the umbilicus the size of a quarter and two dime-sized blebs containing clear fluid on noninflammatory bases located on the upper portion of her chest. Several healing ulcerated areas are present on the back, and two crusted lesions are seen on the nose. Several superficial ulcerations are noted on the lips, tongue, buccal mucosa and soft palate. Her daily temperature has varied between 38.5 and 39.5 C. (101.3 and 103.1 F.).

The urine and the hemogram were normal. The total plasma proteins were 5.97 Gm. per hundred cubic centimeters. The albumin-globulin ratio was 1:10. Hemolytic streptococci and staphylococci were found on culture of material from a bleb. The blood culture was sterile.

Since admission the patient has had innumerable bullous lesions, most of which have ruptured, leaving ulcerated crusted surfaces. The Nikolsky sign is present. Many new and very painful oral lesions have appeared, making it difficult for the patient to eat. She has become somewhat emaciated and extremely toxic.

DISCUSSION

DR. W. R. HUBLER: I should like to make the comment that this case is interesting in that it agrees with the work by Oppenheim (ARCH. DERMAT. & SYPH. **46**:201-206 [Aug.] 1942). He reported 3 cases of pemphigus vulgaris in which there were primary lesions of from one to three months' duration before a generalized eruption appeared. That is true of this case. The primary lesion occurred in the umbilicus and was present for seven or eight weeks before any new lesions appeared.

DR. J. E. FISHER: I had the opportunity of seeing this patient at an early stage of the disease, when she presented a lesion in the umbilicus of three months' duration. At that time there were two lesions in the mouth and one on the arm. I made a diagnosis of pemphigus. Today the involvement of the skin and mucous membrane of the mouth is much more extensive. The prognosis is bad.

DR. R. E. BARNEY: I agree with Dr. Fisher that the prognosis is grave, and I should not be surprised if she were to die in two weeks. I presented a patient about two years ago (ARCH. DERMAT. & SYPH. **44**:128 [July] 1941) whom I treated with germanin. The patient is still free of lesions.

DR. B. F. BARNEY, Columbus, Ohio: About three years ago I had a patient with pemphigus, a woman, whose condition was at least as bad or worse than that of the patient presented today. That was in the early days of therapy with sulfonamide compounds, but nevertheless I advised the administration of large doses of sulfanilamide. While it produced a severe grade of anemia and I had to give blood transfusions every other day, she improved miraculously and was discharged. Since that time, according to the physician who is caring for her, she has had no recurrence of the lesions. She definitely had pemphigus. I feel that in a case like the one presented it will do no harm to push therapy with sulfonamide compounds to top limits in spite of the development of severe anemia.

DR. H. N. COLE: This patient was sent here by Dr. Sidney Littman with the diagnosis of subacute pemphigus. He said that she had a mild form of the disease but did not seem to be doing very well. She was presented before the class of medical students, and after she left the room I remarked that it was a strange disease, that one never could tell what might happen and that in the next week she might present an entirely different picture. In one week her whole picture changed and took on the present appearance.

DR. J. E. RAUSCHKOLB: In speaking of the sulfonamide drugs, the question arises: What does a patient with pemphigus die of, the pemphigus or the secondary superimposed infection? It is known that pemphigus frequently is recurrent. May not a sulfonamide drug clear up the secondary infection instead of acting directly on the pemphigus?

DR. B. F. BARNEY, Columbus, Ohio: In my case I cannot see why there should be such a dramatic regression of the pemphigus after the removal of the secondary infection.

DR. E. W. NETHERTON: I should like to ask Dr. Barney how long his patient has been under observation?

DR. R. E. BARNEY: It has been three years, and there has been no recurrence. Three weeks ago she was alive and well.

DR. C. B. NORRIS, Youngstown, Ohio: I am interested in the point Dr. Barney made as to the use of repeated transfusions in connection with this reaction to a sulfonamide compound; it reminded me of Hollander's method of giving direct transfusions in the treatment of pemphigus. I had a patient, a man, who died about six months ago. I believe that both Dr. Netherton and Dr. LaRocco saw him and concurred in my diagnosis of pemphigus. He lived about nine years after the onset of the disease, during which time he was generally free of the eruption. Recurrences continued in mild form once every six months to a year. Each time there was a suggestion of recurrence he was given direct transfusions of whole blood, 600 cc. at four to six day intervals. I believe this treatment kept him alive all those years. At this particular time I have a patient about 80 years old. No one has verified my diagnosis of pemphigus, but I think I am correct. I have been giving him transfusions by the indirect method, and he has improved. I wonder whether or not there could be any benefit from the transfusions themselves rather than just a cure of the anemia resulting from the sulfonamide drugs?

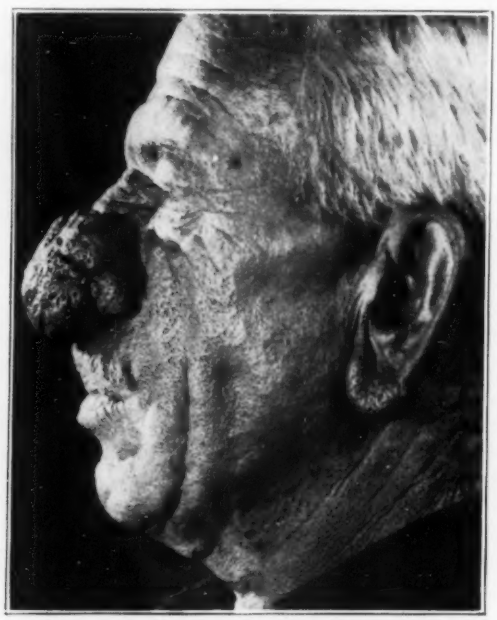
DR. BENJAMIN LEVINE: Last February at the meeting of the Cleveland Dermatological Society at Mount Sinai Hospital I reported on a patient with pemphigus who had died shortly before the meeting (ARCH. DERMAT. & SYPH. **47**:119 [Jan.] 1943). He had been seen in consultation by Dr. Netherton and Dr. Cole. This man had had a great deal of treatment with sulfonamide compounds, particularly with sulfanilamide, up to the point of

toxicity. The levels of the sulfonamide drugs in the blood were watched carefully. His condition became worse with this therapy, the bullous eruption becoming more extensive. He did, however, improve with repeated small blood transfusions of 200 cc. each, given every day or on alternate days. We ran through the whole gamut of therapy with this patient, including administering huge doses of vitamin D, which produced some improvement for a time. Germanin was the only commonly employed drug that was not used. In spite of all the medication and his occasional favorable response to it, he died seven months after the appearance of the first lesion.

DR. E. W. NETHERTON: For several months I have had under observation a woman who was admitted to the hospital with fever, loss of weight, numerous ruptured bullae in the mouth and tense bullae on the skin. When she was admitted she was losing ground rapidly and was toxic. After the administration of large doses of vitamin D she improved and finally became free of lesions. She has remained well as long as she has taken vitamin D. However, when this therapy is stopped, lesions begin to appear in the mouth. There is no one satisfactory treatment for pemphigus. Therapeutic procedures which seem to be beneficial in one case often are of no value in another case of the same disease.

Rhinophyma. Presented by DR. W. R. HUBLER.

J. B., a white man aged 59, presented from the department of dermatology and syphilology, University Hospitals, complains of progressive enlargement of the nose of two years' duration. He has noticed a flushing of the forehead, nose and chin with sudden changes of temperature for about four years. Numerous pinhead-sized pustules have appeared on the same areas. His nose has continued to increase in size and has assumed a permanent reddish color. The patient has consumed from 1 to 3 ounces (30 to 90 cc.) of whisky daily for at least ten years. He uses condiments freely.



Rhinophyma.

The bulbous portion of the nose forms an irregular mass, 6 by 9 by 5 cm., covered by erythematous thickened skin. The flush areas of the face are also red, and the skin over the chin is thickened. A few scattered pinhead-sized pustules are present on the face. The ear lobes are red, enlarged and elongated. The patient has a scrotal tongue.

The urinalysis showed the presence of albumin and many pus cells in a centrifuged specimen. The hemogram was normal, and the Kline reaction negative. A plastic operation on the patient's nose is contemplated.

DISCUSSION

DR. H. N. COLE: This patient is presented preliminary to cosmetic therapy so that the group may see the result afterward.

DR. G. W. BINKLEY: I believe that there are many more patients with rosacea complex among women, especially women in the middle decades, than among men; yet rhinophyma seems to be limited to men. This might point to a different causation for rhinophyma, which has been regarded as the third stage of rosacea.

DR. B. F. BARNEY, Columbus, Ohio: I should like to suggest something to be tried in some cases of this kind. Rhinophyma may be a glandular hypertrophy akin to prostatic hypertrophy. Testosterone may be of value in males and estrogen in females.

Congenital Defect of the Scalp. Presented by DR. GEORGE M. STROUD.

S. S., a girl aged 22 months, presented from the department of dermatology and syphilology, University Hospitals, has had a slowly growing yellowish area of alopecia on the vertex of the scalp since birth.

Just to the left of the vertex of the scalp is a sharply defined, smooth, circular area, about 2 by 2 cm., which is slightly yellow and devoid of hairs or follicular openings. Surrounding it are a few pinhead-sized papules of similar color and consistency with slightly greasy tops. There are no other congenital defects.

The urine and the hemogram were normal. The Kline reaction of the blood was negative. A biopsy was not permitted.

DISCUSSION

DR. H. N. COLE: This is a comman location for a rare condition. In most cases the defect seems to come in the medial line, and in many of them there is this yellowish color. There have been some reports in which even the bone is involved or there is even an absence of bone in the area. Congenital defects on the trunk are occasionally symmetric, like the one described by Sutton (Sutton, R. L., Jr.: Congenital Defects of the Skin in the Newborn, ARCH. DERMAT. & SYPH. **31**:855-857 [June] 1935).

DR. GEORGE M. STROUD: Anderson and Novy (Anderson, N. P., and Novy, F. G.: Congenital Defects of the Scalp, ibid. **46**:257 [Aug.] 1942) reported 4 cases of this disease and mentioned the association with other ectodermal dysplasias. This child, interestingly enough, had another defect in the midline of the body, an umbilical hernia.

Basal Cell Epithelioma of the Scalp with Possible Metastases to the Brain. Presented by DR. W. R. HUBLER.

M. L., a white woman aged 50, presented from the department of dermatology and syphilology, University Hospitals, has had epileptiform attacks for one year. These convulsive episodes have been localized on the right leg. During an attack she is confused and speaks slowly. Her last attack involved the entire right side of the body and resulted in momentary unconsciousness. She has had a chronic ulcer of the scalp for thirteen years. When she was first seen in 1939 it was 8 by 9 cm. in size and roentgenograms revealed an area of bone destruction in its center.

Histologic examination of material from the affected area at that time revealed basal cell epithelioma and chronic inflammation. Roentgenotherapy was employed, 6,000 r being administered to the affected area (two fields). Following this treatment definite healing and filling in of the lesion with epithelial pseudopods were noticed. There remained a 6 by 7 cm. area of exposed skull, which failed to heal in spite of various local applications, including aloes and radon ointments. Recent biopsies of tissue from the edges of the ulcer revealed only granulation tissue and chronic inflammation.

There is an irregular 10 by 10 cm. ulcer which extends in places to the inner table of the skull. Peripheral to this is an irregularly circular area several centimeters wide of alopecia and telangiectasia. The patient has a bilateral Hoffmann sign. She has a weaker grip with her right hand than with her left. The Babinski sign is present on the right side.

The hemogram was normal. The spinal fluid showed 70 leukocytes per cubic millimeter. The Wassermann reaction of the spinal fluid was negative. Roentgenograms of the skull showed destruction of the parietal bones in the region of the ulcer.

DISCUSSION

DR. J. R. DRIVER: This patient has now been followed carefully for three or four years since she was treated. In the light of some of the more recent articles that have been published on effects of irradiation on bone and the deeper tissues, I should be inclined to interpret the

neurologic changes as an effect of irradiation. These convulsive seizures probably result from irritative changes in the meninges and in the brain itself. The area treated was large, the dose was large and because heavy voltage and filtration were used the effects were probably deep. Metastasis is unlikely, since the lesion was of basal cell type. Furthermore, I doubt the existence of malignant growth by direct extension, since there is no evidence of recurrence on the surface. It is well to keep in mind that contrary to older conceptions, bone is one of the more radiosensitive tissues of the body.

DR. H. N. COLE: How would you explain the increased cell count? Were cultures made of the spinal fluid?

DR. W. R. HUBLER: No. I shall repeat the lumbar puncture and get fluid for culture.

DR. J. R. DRIVER: I think the patient has symptoms of meningeal irritation, which would account for the increased cell count.

DR. R. E. BARNEY: She may have a localized encephalitis.

DR. W. R. HUBLER: In a consultation with members of the department of neurosurgery, the possibility was suggested of putting a vitallium plate in the skull in order to remove the focus of irritation. However, it was felt that there was too much infection present on the surface and also that it would be dangerous to attempt this treatment on a patient who had had so much irradiation.

Dermatitis Medicamentosa (Phenolphthalein). Presented by DR. W. R. HUBLER.

L. B., a Jewish boy aged 13, presented from the department of dermatology and syphilology, University Hospitals, was first seen in August 1942, complaining of a generalized "spotty" pruritic eruption which had begun two months before. He gets severe headaches about once a week and for about two months has been taking a tablet of acetylsalicylic acid and 2 ex-lax tablets with each attack. After each such episode he has new "spots" on his skin and the older ones become larger and more pruritic. When he takes only acetylsalicylic acid there is no change noted in his eruption.

There are dime-sized to quarter-sized violaceous macular lesions scattered over the entire body. The lesions are well circumscribed, and some are scaling. The older lesions show only brownish pigmentation.

Twenty-four hours prior to his presentation the patient took ½ ex-lax tablet at my request. Today he presents many new macular erythematous areas, 1 to 3 cm. in diameter, scattered over his body. Each of the pigmented areas remaining from the previous eruption is surrounded by an erythematous halo.

DISCUSSION

DR. R. E. BARNEY: In addition to this interestingly demonstrated eruption due to phenolphthalein, this boy has a patch of gray hair about the size of a quarter, which his mother said had been present since birth. It is similar to the type one occasionally sees after alopecia areata in adults when the hair sometimes comes in gray. Since this process has been present since birth, it probably is a nevus.

Sulfathiazole Eruption. Presented by DR. GEORGE M. STROUD.

F. H., a white man aged 37, presented from the department of dermatology and syphilology, University Hospitals, has exhibited repeated cutaneous sensitivity to sulfathiazole. A year ago he was shown before this society with a penile chancre representing a probable reinfection in congenital syphilis (ARCH. DERMAT. & SYPH. **45**:827 [April] 1942). This diagnosis was finally made when the administration of mapharsen caused rapid healing. During the time of investigation of this case in which repeated dark field examinations for Spirochaeta pallida and serologic tests of the blood for syphilis gave negative or very weakly positive results, a therapeutic trial of orally administered sulfathiazole resulted on the tenth day in the appearance of a fairly generalized erythema, conjunctivitis, chills, fever, nausea and vomiting. A prompt recurrence of these symptoms as well as swelling of the penis and inguinal lymph nodes occurred when the powdered drug alone was applied locally ten days later. Orally administered sulfanilamide was then used for five days, but a fever, with a temperature of 39.2 C. (102.6 F.), suddenly developed. The temperature became normal the day after administration of this drug was discontinued.

Two months ago the patient was readmitted to the hospital, and at 9:30 a. m. 20 mg. of sulfathiazole was given orally without reaction. Two grams of the drug was given at 11:45 a. m. and 1 Gm. at 4:00 p. m. Within four hours chilliness, fever (temperature of 39.4 C. [102.9 F.]), conjunctival injection and erythema of the head, neck, shoulders and trunk developed. Reactions to patch and precipitation tests with suspensions of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine were negative at this time. At 12:30 p. m. today,

three hours before presentation, he was given orally 3.5 Gm. of sulfathiazole. Within two and one-half hours he had nausea, malaise, fever, chilliness, conjunctival injection and erythema on the face, trunk and arms.

DISCUSSION

DR. GEORGE M. STROUD: I have investigated this case in an attempt to determine whether patients who once have had sulfathiazole are going to become sensitive during a subsequent course. It is true that some patients given the drug for pneumonia show no reaction but in subsequent courses present evidence of intolerance. Wedum (*J. Infect. Dis.* **70**:173, 1942) reported on the inability to produce sensitivity to sulfonamide drugs in animals, as shown by cutaneous tests, precipitins and anaphylaxis. By combining these drugs with serum and producing an azoprotein he was able to sensitize the animals by the aforementioned methods. I shall attempt to develop such a protein, which might be used in determining human sensitivity by means of a cutaneous or precipitin test. Such a method would certainly be much less harmful than the use of a "test dose," which is the only one in present use.

Opaque Cornea Due to Interstitial Keratitis Treated by Keratoplasty. Presented by DR. GEORGE M. STROUD.

L. K., a white man aged 33, presented from the department of dermatology and syphilology, University Hospitals, was first seen in 1936 because of poor vision. He had been treated elsewhere with injections in the arm and hip for thirteen weeks, without improvement. Vision in the right eye was limited to perception of light and in the left to recognition of fingers at a distance of 2 inches (5 cm.). A diagnosis of congenital syphilis with active syphilitic interstitial keratitis and myopia was made, and he was given twelve satisfactory febrile paroxysms of malaria.

During the next four years he received fairly regular alternating courses of mapharsen and bismuth. The keratitis healed, leaving such opacities that vision was still reduced to recognition of hand movements in the right eye and to 6/60 in the left. Optic iridectomies performed in 1938 did not improve his vision. For this reason he had a full thickness keratoplasty of the right eye performed seven months ago using a square graft according to the technic of Castroviejo. Within three weeks the graft became edematous and mottled and circumcorneal injection developed. Reinoculation with malarial blood was unsuccessful, and the patient was then given six satisfactory febrile paroxysms, with typhoid vaccine given intravenously. The graft became clear and the circumcorneal injection minimal within six weeks. No chemotherapy has been given.

At present there is a square scar on the right cornea with a slight opacity peripherally. There are diffuse deep corneal nebulae on the left cornea. There is an iridectomy defect in each eye. Vision in the right eye is 6/20 after compensation for the myopia.

The Kline reaction of the blood a month ago was slightly positive. A keratoplasty on his left eye is being considered.

DISCUSSION

DR. C. L. CUMMER: Was this patient supposed to have acquired or congenital syphilis?

DR. GEORGE M. STROUD: He has proved congenital syphilis.

DR. C. L. CUMMER: I think a case like this is of extreme interest because of the late appearance of interstitial keratitis. A number of years ago I took care of a woman aged 26 who had severe interstitial keratitis after the birth of her first child. The child had a negative Wassermann reaction. Despite the institution of early treatment the keratitis went on and caused permanent and complete scarring of both corneas, resulting in almost total blindness. This case also illustrates the late stage at which interstitial keratitis can appear. The result of keratoplasty in the case presented today is most remarkable. It is almost a miracle to have restored the vision to this man's eyes.

DR. J. R. DRIVER: Of interest, too, is the fact that this patient had a relapse of his interstitial keratitis a few weeks after the keratoplasty. Fortunately the response to further fever therapy was satisfactory, and the final result of the operation was most gratifying. The factor of trauma in causing relapses of this type should be kept in mind when keratoplasties are performed on syphilitic patients.

Argyll Robertson Pupils Occurring in Neuromyelopathy Simulating Tabes Dorsalis. Presented by DR. GEORGE M. STROUD.

C. L., a white man agd 48, presented from the department of dermatology and syphilology, University Hospitals, through the courtesy of the department of medicine, has had an ulcer on the left sole for four years. He had known and poorly controlled diabetes for eleven years.

He has had weakness and diminished to no sensation in the extremities, ataxia and loss of weight in the past four years. Visual acuity has diminished moderately in the past year. He has consumed on an average between a pint and a quart (500 to 1,000 cc.) of whisky daily for the past twenty years. He gives no personal or familial history of syphilis. He has not had gonorrhea. In his work, during the past few years, he has traumatized his legs frequently, with resultant scarring and brown pigmentation. During the past few months he has had partial urinary incontinence.

The man is poorly nourished. The skin is dry, and on the legs there are thick scales and crusts, most prominent distally. Over the tibias are large brown macules with some atrophy centrally. The nails are thickened and deformed. Near the center of the left sole is a fairly deep, clean, punched-out defect, which is thinly epithelized. The pupils are small and irregular, and the left is larger than the right. They do not react to light but do react in accommodation. The ciliospinal reflex is absent. The legs are thin and weak and are without tendon reflexes. Sensation is diminished to absent over the distal parts of the extremities. There is no penile scar. The posterior tibial and dorsalis pedis arteries are not palpable.

There were moderate pyuria and slight glycosuria. The hemogram was normal except for an erythrocyte count of 3,000,000 per cubic millimeter and a hemoglobin content of 60 per cent. The mean corpuscular hemoglobin concentration was 29 per cent. The Kline and Wassermann reactions were negative. The spinal fluid was normal save for a protein content of 93 mg. per hundred cubic centimeters. Free acid was present in the gastric juice. The blood sugar was 186 mg. per hundred cubic centimeters.

By control of his diabetes, use of a good diet, abstinence from alcohol, rest in bed and the administration of brewer's yeast, nicotinic acid and thiamine hydrochloride, the patient's vision, sensation, strength and plantar ulcer have improved. Cod liver oil ointment has been applied to the ulcer.

DISCUSSION

DR. R. E. BARNEY: There must be decided changes in this man's central nervous system. Twenty years of alcohol addiction, with drinking of up to a quart a day, should do something to the nervous system. He has atrophy of the small muscles of the hand, in addition to what appears to be a trophic ulcer of the left sole. The little toe appears to be held by only a small pedicle. This, according to the patient, is due to the bone being destroyed by a carbuncle.

Anergic Proliferative Tuberculosis of the Skin. Presented by DR. CLAUDE B. NORRIS, Youngstown, Ohio.

H. S., a Negress aged 51, has had a lesion on the face which has slowly increased in size over a period of thirty-five years. She complains of no subjective symptoms.

There are thickened and raised lesions involving the borders of the lobe of the right ear and extending downward in an irregular form almost to the chin and back of the ear about 1 cm. It then extends forward over the bridge of the nose, involving the lids of both eyes and surrounding structures, and to within 1.5 cm. of the left ear. The left ear is involved with nodular thickening, as is the case with various portions of the lesions on the face. There is complete alopecia of the brows and most of the lashes. The forehead shows reddish depigmentation of a considerable area. There is surprisingly little scar formation and no ulceration. There are a nodule on the left arm and one on the right thigh, presenting a similar appearance.

The Kahn and Kline reactions were negative. Histologic examination showed subepithelial infiltration of granulomatous tissue within which there were many discrete tubercles containing Langhans giant cells. There was plasma cell and histiocyte infiltration about the tubercles. There was no caseation in the tubercles, so that the picture was that of anergic tuberculosis, which has sometimes been confused with Boeck's sarcoid.

DISCUSSION

DR. E. J. ARDAY: Does anergic refer to the fact that the patient does not react to the specific immunologic substance, i. e., tuberculin?

DR. C. B. NORRIS, Youngstown, Ohio: The diagnosis is that of the hospital pathologist, based in part, I think, on the history. The lesion has been present thirty-five years, indicating anergy. A tuberculin test was not done, nor were roentgenograms of the chest and bones made.

DR. J. R. DRIVER: I, too, think this patient has lupus vulgaris. Has she received any treatment?

DR. C. B. NORRIS, Youngstown, Ohio: She has received no specific treatment. Salves of various kinds have been employed. I administered a salt-free, high caloric diet and a combined vitamin preparation that contains a large amount of vitamins A, D and B complex. I have had her take rather heavy doses of it; she is apparently getting some effect from the nicotinic acid, because soon after she takes the preparation she gets flushing, that has resulted in some desquamation and a burning sensation, which she did not have before.

NOTE.—Dr. Norris later reported that roentgenograms of the chest showed no evidence of pulmonary tuberculosis or other disease in the lungs. Vollmer's tuberculin patch test was performed on the forearm, where the patch remained for forty-eight hours. The reading of the test was done forty-eight hours after the patch was removed, i. e. four days after the patch was first put on. It showed a sharply circumscribed, slightly infiltrated reddened square with lichenoid-follicular elevations.

Sarcoidosis. Presented by DR. GEORGE M. STROUD.

C. W., a Negress aged 25, presented from the department of dermatology and syphilology, University Hospitals, has had a nodular eruption on the legs for the past four months. She has been followed in the ophthalmic department of the hospital for two years because of a recurrent iridocyclitis. Seven months ago she had bilateral swelling of the parotid glands, which was associated with pain on eating most foods, particularly salty ones. The eruption on the legs and the parotid swelling have recently partially subsided. There is no history of tuberculosis in the patient or in her family. She has lost a slight amount of weight.

The right eye is normal except for a few keratic precipitates centrally and below. The left eye shows a mild circumcorneal congestion. The pupil is well dilated with a slight vitreous haze and mild hyperemia of the disk. Slit lamp examination showed increased protein and cells in the aqueous humor and a few small keratic precipitates. There is slight bilateral swelling in the region of the parotid glands. There is a moderately indurated well defined papular eruption on the thighs. There are a few discrete indurated nodules and scaling on the legs. There is no significant peripheral adenopathy. She has had a moderate elevation of temperature during her stay in the hospital.

The hemogram and urine were normal. Reactions to serologic tests for syphilis were negative. The albumin content of the blood was 3.4 Gm. and the globulin 2.8 Gm. per hundred cubic centimeters. The albumin-globulin ratio was 1:2. The sedimentation rate was slightly accelerated. There was a moderately positive reaction to the tuberculin test with the 1:10 dilution. Roentgenograms showed no abnormality in the pulmonary fields except slight enlargement of the right paratracheal lymph node. Roentgenograms of the fingers showed no abnormality.

Histologic examination of a nodule on the leg revealed a noncaseating granulomatous infiltrate in the corium consisting of giant cells and tubercles.

DISCUSSION

DR. C. L. CUMMER: I cannot see on what grounds the presenter can sustain the diagnosis of sarcoidosis. The lesions are gone. Roentgenograms of the chest seem to show no pathologic changes. I should like to hear the logic in this diagnosis.

DR. J. E. RAUSCHKOLB: I think the clinical picture fits in with erythema induratum. She has the residual of a recurrent conjunctivitis. She has a small, almost lima bean–sized adenopathy in the cervical area. And the nodules on both legs, while there was no ulceration, certainly fit the clinical features of erythema induratum rather than those of sarcoidosis.

DR. H. N. COLE: I agree with Dr. Rauschkolb that the lesions on the calves are more suggestive of erythema induratum than of sarcoidosis. However, the history and the rest of the picture are rather typical of Heerfordt's disease, a type of sarcoidosis. She has had parotitis. She has the ocular involvement and she has an anergy, all going with Heerfordt's disease, and I do not think it is impossible for this woman to have sarcoidosis and also tuberculosis. She may have a borderline condition, for the causative organism in sarcoidosis, according to Schaumann, is an anergic strain of tuberculosis.

DR. GEORGE M. STROUD: The relatively superficial character of the papules on the thighs, the absence of caseation clinically or histologically, the coexistence of uveoparotitis and the relative anergy to tuberculin lead me to put this eruption in the category of sarcoidosis of the erythema induratum-like type.

In reference to the conditions which are borderline between frank tuberculosis and sarcoidosis, I should like to mention a case of classic sarcoidosis (Hollister, W. F., and Harrell, G. T.: Generalized Sarcoidosis of Boeck Accompanied with Tuberculosis and Streptococcic Bacteremia, *Arch. Path.* **31**:178 [Feb.] 1941) in which caseating pulmonary tuberculosis was found at postmortem examination.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

FRANK C. COMBES, M.D., *Chairman*

PAUL GROSS, M.D., *Secretary*

Nov. 4, 1942

A Case for Diagnosis (Lichen Spinulosus? Lichen Planopilaris?). Presented by DR. VAN ALSTYNE H. CORNELL.

B. L., a woman aged 60, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital with an eruption which has been present for six weeks.

On both shoulders, the lateral parts of the back and buttocks, the lateral aspects of the chest, the extensor surfaces of the arms, the lateral aspects of the thighs, the breasts and the abdomen there is a pronounced follicular eruption of tiny skin-colored papules. In some areas, as on the shoulders, typical horny spines emerge from the follicular openings. On palpation the surface in these areas feels like a nutmeg grater. At times there is severe itching, usually in the evenings. There are no lesions in the mouth.

The Wassermann and Kahn reactions of the blood were negative, and a complete blood count was normal.

The histologic examination revealed lichen planopilaris.

DISCUSSION

DR. DAVID BLOOM: Clinically the case is one of lichen spinulosus. It seems to me that the histologic picture of so-called lichen planopilaris is present in every case of lichen spinulosus. I am therefore inclined to consider that this pathologic picture does not necessarily indicate the presence of lichen planus.

DR. WILBERT SACHS: It is seriously doubted whether lichen spinulosus exists in the adult. This is a characteristic case of lichen planopilaris. The patient will probably have lesions of lichen planus later on. One cannot disregard the histopathologic picture. The patient showed a definite picture of lichen planus of the hair follicles.

Lichen Sclerosus et Atrophicus. Presented by DR. TIMOTHY J. RIORDAN.

P. K., a woman aged 52, presented from Bellevue Hospital, noted a year ago for the first time a few small papules on her left wrist, which slowly increased in size. Similar papules then appeared on the right wrist and the left knee.

On both wrists and elbows and on the left knee there are whitish, irregular plaques consisting of pea-sized, slightly scaling papules which show comedo-like centers.

The Wassermann reaction of the blood was negative; the blood and urine were normal, and a roentgen examination of the chest revealed no abnormalities.

The histologic picture was consistent with the clinical diagnosis of lichen sclerosus et atrophicus.

The patient has received several injections of a bismuth compound and local applications of boric acid ointment.

A Case for Diagnosis (Dermatitis Herpetiformis? Erythema Multiforme? Pemphigus?). Presented by DR. ISADORE ROSEN.

A. B., a man aged 49, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital with a generalized eruption of a year's duration.

On the face, scalp, trunk and upper and lower extremities the patient presents many coin-sized raised crusted lesions and a few bullae. Itching is moderate. The scalp shows diffuse crusting. There are no lesions on the mucous membranes of the mouth.

The past history and family history are irrelevant.

The Wassermann and Kahn reactions of the blood were negative. The complete blood count was normal except for 8 per cent eosinophils. The serum from a bulla showed 20 per cent eosinophils. Chemical examination of the blood showed 26 mg. of nonprotein nitrogen and 510 mg. of sodium chloride per hundred cubic centimeters. The histologic diagnosis was pemphigus. A patch test with 20 per cent potassium iodide ointment resulted in a mild vesicular reaction in normal skin, with a more severe reaction in the affected skin. The reaction to a control test with petrolatum was negative. The ingestion of potassium iodide, 0.5 Gm., produced numerous erythematous plaques covered with grouped vesicles or bullae.

The patient is receiving sodium arsenate, 2 per cent solution, subcutaneously.

DISCUSSION

DR. DAVID BLOOM: In spite of this patient's strong sensitivity to potassium iodide, I favor the diagnosis of pemphigus vulgaris rather than dermatitis herpetiformis.

DR. WILBERT SACHS: It is true that the bullous lesions of pemphigus can be epidermic and sometimes subepidermic. This lesion was epidermic and therefore tends much more to resemble pemphigus and to rule out dermatitis herpetiformis. Other features of the slide suggested pemphigus.

DR. MAURICE J. COSTELLO: I should like to ask whether sulfathiazole or sulfapyridine was used as a therapeutic test. I think it is fairly well established that most patients with dermatitis herpetiformis respond favorably while the drug is being administered.

DR. E. WILLIAM ABRAMOWITZ: When I examined this man for the first time I thought he had pemphigus. The next time I observed him he presented gyrate erythematous patches; so I changed my diagnosis to dermatitis herpetiformis or possibly erythema multiforme. The potassium iodide test has to be studied further to convince every one that it is reliable. I place more dependence on the presence of the Nikolsky sign in cases of this sort, although it may appear with other bullous dermatoses. In this particular patient the sign was positive; hence he may have pemphigus after all. He received iodides and a bullous eruption developed so that he now has a drug eruption.

DR. PAUL E. BECHET: I have so frequently observed Nikolsky's sign in bullous dermatoses other than pemphigus that I do not believe it is of any diagnostic value whatsoever in pemphigus.

DR. PAUL GROSS: There are cases of bullous eruption which are difficult to classify. I have not been inclined to accept cases in which there are large bullous lesions as instances of dermatitis herpetiformis. The diagnosis of erythema multiforme bullosum is frequently questioned on account of the presence of numerous bullae on normal skin.

DR. FRANK C. COMBES: There are several points against a diagnosis of pemphigus, one of the most important being that no lesions are found on the mucosa; this is unusual considering the extensive involvement. Secondly, in spite of this extensive involvement, the patient seems to be in good health, with no loss of weight. Thirdly, many of the bullae are irregular or stellate in shape, which is unusual in pemphigus but common in dermatitis herpetiformis and erythema multiforme.

DR. ISADORE ROSEN: I have never seen so extensive a reaction following the potassium iodide test. In this case, as a result of the reaction, one would be more inclined to make a diagnosis of pemphigus than of dermatitis herpetiformis or erythema multiforme. Before the administration of potassium iodide the lesions were dry with an erythematous base surrounded with pigmentation and accompanied by severe itching. At no time have there been any lesions in the mouth. The reason that there have not been more positive reactions following the local application of potassium iodide in ointment form is that the importance of freshly prepared ointment thoroughly incorporated in the making has not been stressed.

A Case for Diagnosis (Multiple Benign Cystic Epithelioma? Neurofibroma of the Skin?). Presented by DR. MAX SCHEER.

B. G., a woman aged 59, registered at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on Oct. 29, 1942, presenting growths of twenty-five years' duration. She had two miscarriages prior to the birth of her children. No

definite history could be elicited of any similar disease in her two daughters or distant relatives. In 1928 she had a hysterectomy. In 1929 a plum-sized fatty tumor was removed from the back. A small growth was removed from the right elbow joint in the New York Skin and Cancer Hospital in 1930. Her general health has been good since.

The growths began in the nasolabial groove and spread to the forehead. About a year later she noticed growths on her arms. She has observed that during the past year some of the nodules have become smaller and two lesions on the left cheek have disappeared.

The growths are limited to one side of the face and are located on the forehead, in the nasolabial groove and at the left side of the mouth. There are isolated lesions on the left side of the neck and the outer surfaces of the left hand and forearm and to a lesser extent on the right hand. There are also isolated nodules on the back and some grouped lesions on the upper part of the breasts. There are no growths on the abdomen or lower limbs.

The lesions are skin colored, discrete, soft and raised, varying in size from that of a pinhead to that of pea. Some give the suggestion of slight herniation. They are easily compressible and with a diascopic glass reveal a light bluish to light brown stain. On the left inner side of the eyebrow is a hard, fibrotic, small grape-sized growth. There are several café au lait spots on the left side of the face and on the left breast.

DISCUSSION

DR. CHARLES LERNER: My diagnosis is von Recklinghausen's disease. The lesions are too large and do not possess the glistening appearance that often goes with multiple benign cystic epithelioma. They also have a doughy feeling such as one gets on palpating von Recklinghausen's tumors.

DR. MAX SCHEER: I made a diagnosis of neurofibroma. There were compressible tumors, and, although they were small, I could feel in several of them a slight herniation; there were also some café au lait spots. However, some of my colleagues who examined the patient made a diagnosis of multiple benign cystic epithelioma. I did not agree with them. The histologic report is not yet ready.

A Case for Diagnosis (Syphilitic Juxta-Articular Nodes?). Presented by DR. NATHAN SOBEL.

C. G., a Negro aged 47, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital. Below each elbow there is a walnut-sized, fairly firm nodule which is not attached to the skin or underlying tissue. The patient states that he received a few intravenous injections for arthritis several years ago. Arthritis of the finger joints is present.

The Wassermann and Kahn reactions of the blood were negative.

Biopsy revealed numerous small blood vessels throughout the tissue. About these was a tremendous increase in fibrous tissue with a diffuse fibroblast cell infiltration. In one section was an entire area which took a more or less homogeneous stain, indicating calcium. In one or two areas there were small collections of large multinucleated cells, probably foreign body giant cells. There were one or two small areas of necrosis. There were no noteworthy changes in or about the vessels. There were no plasma cells. The diagnosis was granulation tissue with foreign body giant cell reaction and deposition of calcium (Dr. Wilbert Sachs).

A course of injections of a bismuth compound has resulted in considerable diminution in size of the lesions.

DISCUSSION

DR. WILBERT SACHS: In nodes of this type one should find some evidence of syphilis, such as fibrosis, vascular changes and a plasma cell reaction. These are juxta-articular nodes, but here one finds calcium deposit, foreign body giant cells and no vascular changes. Therefore I believe the disease is not due to syphilis.

DR. ISADORE ROSEN: On clinical grounds alone one is not really entitled to accept that diagnosis, but the fact that a decided improvement followed a few injections of a bismuth compound would suggest that the process was of syphilitic origin.

DR. NATHAN SOBEL: This man has responded to treatment, as Dr. Rosen said, and the nodes are much softer and smaller. That would favor the diagnosis, because response to antisyphilitic therapy has been often reported. As far as calcium deposits are concerned, it is rare to find them within the nodes. In regard to the Wassermann reaction, quite a few of the patients with this condition have negative reactions, just as do persons with other forms of late syphilis, although in a majority of cases the reaction is positive.

LOS ANGELES DERMATOLOGICAL SOCIETY

L. F. X. WILHELM, M.D., *President*

ARTHUR FLETCHER HALL, M.D., *Secretary*

Nov. 11, 1942

Malignant Reticuloendothelioma. Presented by DR. IRVING BANCROFT and DR. JOSEPH MIROVICH (by invitation).

L. M., a 69 year old white housewife, had no eruption until eight or nine months ago, when she noticed a purplish spot on the right leg just above the ankle. This lesion enlarged peripherally, and later several new lesions appeared in a line toward the groin. There is now a confluent mass of tumors covering the lower part of the right leg. This has been formed by bright red patches which have become tumors, gradually enlarging to become confluent. The mass thus formed is bright red, warm to the touch, nontender and nonpruritic.

The results of examination of the blood were as follows: The Wassermann and Kahn reactions were negative. The nonprotein nitrogen content was 45 mg. and the iodine content 4.9 micrograms per hundred cubic centimeters. Tests showed no bromine. The hemoglobin content was 94 per cent; the erythrocytes numbered 5,350,000, and the color index was 0.94. The total leukocytes numbered 7,500, of which 62.5 per cent were polymorphonuclear leukocytes, 27 per cent small lymphocytes, 9.5 per cent large mononuclear cells, 0.5 per cent eosinophils and 0.5 per cent basophils.

The urine was normal. Roentgenograms of the chest showed no evidence of neoplasm. A biopsy suggested reticuloendothelioma with some features not unlike monocytic leukemia.

Various areas have received from 100 to 300 r of roentgen rays, filtered through 0.25 mm. of copper; this treatment has been followed by decided regression in the size of the tumors. Those areas treated first (two weeks before this meeting) and receiving 200 r, total dosage, show 75 per cent improvement.

DISCUSSION

DR. NELSON PAUL ANDERSON: The response to roentgen rays has been remarkable. I still do not know in what category to place this lesion. Histologically, when it was first seen I made a diagnosis of reticuloendothelioma. I believe the response to radiation has been such that one can more or less exclude Kaposi's sarcoma. I wonder if Dr. Newton Evans, in the department of pathology of the Los Angeles General Hospital, has seen the slides.

DR. JOSEPH MIROVICH (by invitation): Dr. Evans studied the slides and said that he believed they showed mycosis fungoides.

DR. H. P. JACOBSON: This patient was presented before the Malignancy Board last week. Dr. Newton Evans, a pathologist and a member of the board, reviewed the slides and expressed the opinion that the microscopic architecture was consistent with mycosis fungoides. From the purely clinical standpoint, the picture suggests the possibility of leukemia. A reticulum cell sarcoma, especially in the light of Dr. Anderson's microscopic interpretation, could not be eliminated with any degree of certainty.

LIEUTENANT COMMANDER LEE MCCARTHY (MC), U.S.N.R. (by invitation): I think everybody ought at least to express an opinion on such an interesting case. I believe this condition is mycosis fungoides. It is unusual to have the lesions limited to the leg, but on the thigh there is one lesion showing typical configuration of mycosis fungoides. Although the section is cut a little thick, I thought I found all the cells usually seen in mycosis fungoides. There were quite a few plasma cells, one or two giant plasma cells and quite a number of mycotic or large cells. It might be mycosis fungoides d'emblée, which has no prodromal signs but only tumors.

DR. JOSEPH MIROVICH (by invitation): The blood picture was normal.

DR. W. H. GOECKERMAN: It is difficult to go against the somewhat confusing histologic picture. From the clinical aspects I thought there must be leukemic infiltration. The normal blood picture does not entirely militate against that.

Epidermolysis Bullosa. Presented by DR. ANKER K. JENSEN.

G. C., a 16 year old white school girl, has suffered from a bullous eruption of the skin since birth; her familial history and past history, except for her cutaneous disease, tells nothing of interest. At present she shows numerous old scars on the extremities, body and face. There are a large area of granulation tissue on the back of the neck and one on the flexor surface

of the right arm. The Wassermann reaction was negative, and urinalysis gave normal results. A biopsy, made at the Los Angeles County Hospital, showed a picture consistent with epidermolysis bullosa. She has been treated with a high vitamin diet and with roentgen rays (600 r to the granulation tissue on the back of the neck and 450 r to that on her arm).

DISCUSSION

DR. KENDAL FROST: I agree with the diagnosis. There are one or two points that are rather confusing to me. To see her arms alone, it looks almost as though those lesions were neurotic excoriations. On the nape of her neck is this long, granulomatous, elevated lesion, which I am at a loss to explain. It has been there for several years. Whether this is a keloid kept open or a neurotic excoriation which she might produce herself, I do not know.

DR. C. R. HALLORAN: I was impressed, as was Dr. Frost, with the element of hypertrophic tissue that looks keloidal, especially the lesion on the neck. However, on one elbow there is a stretched type of scar typical of epidermolysis bullosa.

DR. NELSON PAUL ANDERSON: I think Dr. Jensen is to be congratulated for presenting this case. It is one of the cases all the members have encountered at the Los Angeles County Hospital, and I do not believe there is a doubt as to the diagnosis. I wish Dr. Jensen would continue to present this patient from year to year, because one can look over the literature on epidermolysis bullosa and not find the outcome in a single case. Apparently this case is going to furnish Dr. Jensen with the opportunity of following it for a long time. I can remember when the patient had a single lesion on the nasolabial fold and a lesion on the upper eyelid. Tonight she presents a little scarring from the lesion on the nasolabial fold. Incidentally, when I saw this girl five or six years ago she had practically no teeth. Since she has had all her teeth extracted and has had artificial dentures made, it has improved her appearance and has considerably improved her general nutrition. I think that the lesion on the back of the neck can be treated by taking a sharp curet and removing the excessive granulations clear down to firm fibrous tissue. This should give it a good chance of healing.

DR. M. E. OBERMAYER: I fully agree with Dr. Anderson as to the desirability of a systematic follow-up study of patients with this disease. The situation in epidermolysis bullosa is similar to that in chronic lupus erythematosus; in both dermatoses one rarely knows the eventual outcome of the disease. I suggest that studies for porphyria be made. Turner and I, and others, have reported the association of acquired epidermolysis bullosa with porphyria, melanosis and hypertrichosis; though I did not believe that the porphyria was causative, it may be that studies in this direction will eventually yield etiologic contributions of significance.

DR. ANKER K. JENSEN: I knew most of the members had seen this girl before, a number of years ago, and I thought they might be interested in knowing what progress she is making. The chief difficulty now is the two large areas of granulation tissue, one on the back of the neck and one on the right arm. These lesions were cauterized after the curettement of the granulation tissue. But it was not long until they had recurred again. Pressure bandages have also been tried, with no success. At present she is using a powdered burnt alum.

Dermatitis Venenata and Lupus Erythematosus. Presented by DR. ANKER K. JENSEN.

C. M., a 57 year old white man, has had attacks similar to the present one for the past two years. The lesions come and go. When first seen this patient presented an erythematovesicular, slightly desquamating eruption on the forehead. It became worse when he perspired. There is also a well marginated purplish red plaque the size of a 50 cent piece on the right temple. This patch is itchy and slightly scaly. It seems to be somewhat indurated but not elevated above the surface.

There is no history of the ingestion of any drugs except magnesia magma. The Wassermann reaction of the blood was negative a few months ago, and the urine was normal. The reaction to a patch test with his hat band was negative. A section of tissue is presented for microscopic examination. He has received a bismuth compound intramuscularly and a few fractional doses of roentgen rays locally.

DISCUSSION

DR. SAMUEL AYRES: I do not see any evidence of lupus erythematosus now. I did not see the slide, but the eruption that the patient shows is more of a contact dermatitis. He works with oil and grease. He also uses a proprietary hair oil. That might possibly have some bearing on it.

DR. KENDAL FROST: There is a plaque over his right zygoma which I thought was lupus erythematosus. It is a combination one does not often see.

DR. C. R. HALLORAN: I agree with the diagnosis of lupus erythematosus. The lesions on the forehead I saw in a pretty good light. Clinically they resemble lupus erythematosus. I do not see any dermatitis venenata now. I understand that this eruption appears as a dermatitis venenata and when the acute lesions are cleared they appear as lupus erythematosus.

DR. SAUL ROBINSON: I believe this patient has lupus erythematosus, and I understand that a bismuth compound is being given now. It may be that treatment with a gold compound or freezing with solid carbon dioxide will clear up the patch over the zygoma.

DR. ANKER K. JENSEN: When I first saw this man he presented a vesiculoerythematous eruption conforming closely to the hat band area. There was also a violaceous indurated plaque on the right temple. The lesions on his forehead were due to the hat band, and the one on the side of his face is lupus erythematosus.

A Case for Diagnosis (Syphilis Simulating Lupus Erythematosus?). Presented by DR. H. C. L. LINDSAY.

H. T., a 24 year old Negress, was first found to have syphilis when she was four months pregnant, in August 1936; she had had a miscarriage three years previously. During the next six years she received antisyphilitic treatments irregularly, and the Wassermann reaction of the blood varied between 1 plus and 4 plus; the spinal fluid was normal. On Dec. 31, 1941 she presented herself with a lesion on the vermilion border of the right side of the upper lip, which looked like a "mucous patch" of syphilis, and a lesion on the left malar region, of four months' duration, which closely resembled lupus erythematosus clinically. The Kolmer reaction was 3 plus at this time. During the next three months the cutaneous lesion improved under treatment with bismuth arsphenamine sulfonate, seven doses of 0.1 Gm. each, intramuscularly. Four months later, however, a plaque appeared symmetrically situated on the opposite side, as well as a small similar lesion above the right eyebrow. These lesions she shows tonight; a scant scale is present, which is not strongly adherent, and no follicular plugging is seen. The blood count was normal. Considerable improvement has followed the administration of sulfanilamide, 1 Gm. daily by mouth, and the local use of sulfathiazole ointment; the patient has had a total of thirty-six injections of arsenicals and sixty-seven of a bismuth compound, somewhat irregularly.

DISCUSSION

DR. SAUL ROBINSON: I think I would change the diagnosis to "lupus erythematosus simulating syphilitic lesions."

DR. IRVING BANCROFT: I should call the patient's condition "lupus erythematosus and latent syphilis."

DR. C. R. HALLORAN: I agree with the diagnosis of lupus erythematosus. I am not much in favor of the use of neoarsphenamine for this disease. I have had a patient at the Los Angeles General Hospital, whom you have all seen, with a resistant lupus erythematosus of the scalp. I gave him neoarsphenamine, and there was a dissemination of his condition.

DR. KENDAL FROST: I encountered a case in point several years ago. No biopsies were made, but there was a positive serologic reaction for syphilis. Clinically, the lesion was lupus erythematosus. When the patient was treated for syphilis only, it took the lesion about six months to clear, but it cleared completely. It was much too slow for specific therapeutic response to antisyphilitic treatment. The lesion was clinically one of lupus erythematosus producing deep scarring.

LIEUTENANT COMMANDER LEE MCCARTHY (MC), U.S.N.R. (by invitation): It is not unusual to see concomitant lesions on the scalp in this race.

DR. SAMUEL AYRES: Clinically the lesion looks more like lupus erythematosus than syphilis, although the case reminds me of one I observed several years ago, that of a Negro boy, in which the diagnosis was lupus erythematosus. A positive serologic reaction of the blood for syphilis was found, and the cutaneous lesion healed rapidly under antisyphilitic treatment. It was also typical of lupus erythematosus.

DR. W. H. GOECKERMAN: I agree with the diagnosis of lupus erythematosus. Before the gold treatment was established, and arsphenamine therapy was in its infancy, I tried it in a fair number of cases of lupus erythematosus, and in most of them the condition was made worse. If this patient's lesions respond to arsphenamine, I shall change my diagnosis to syphilis.

DR. H. C. L. LINDSAY: I have looked at this patient for a long time. Sometimes the lesions look more like lupus erythematosus than otherwise. I consider that some of the fine points necessary in making a diagnosis of lupus erythematosus in this patient's case cannot be demonstrated clinically, and my diagnosis is "syphilis lesions simulating lupus erythematosus."

Some French dermatologists used neoarsphenamine in the treatment of lupus erythematosus for a long time after gold was so favorably endorsed. Two years ago a patient, well known to most dermatologists in Los Angeles and to many in San Francisco, came to me for treatment of lupus erythematosus. During twelve years she had intermittently been treated with gold sodium thiosulfate and a bismuth compound and locally with solid carbon dioxide. The Wassermann test had been repeatedly made. A further check of her condition indicated that she did not have syphilis. Nevertheless, her response to neoarsphenamine was rapid and complete. This patient had lupus erythematosus which responded to antisyphilitic treatment. It shows the fallacy of the "therapeutic test" in syphilis.

Tertiary Syphilis. Presented by DR. IRVING BANCROFT.

P. V. D., a 43 year old white man, had many tests of the blood for syphilis before 1929, all of which gave negative reactions. He gives no history of a primary sore or rash, except an eruption due to poison oak, which lasted a few days only. The present eruption started about three years ago. The lesions are mostly on the right forearm and more especially on the extensor surface and below the elbow. They consist of slightly raised papules, mostly irregular or circular and about ½ inch (1.3 cm.) in diameter. Many are fused into plaques and in some places are covered with scales and in others are crepelike on the surface. The color is bright red except that some of the flatter lesions are paler. Some are slightly raised, and others are level with the surface of the skin. There is another group of lesions over the left shoulder blade which are pale pink and which present a slightly circular shape. The Wassermann reaction was positive at the time of the Selective Service examination and twice since, during the last few weeks. He has received two injections of a bismuth compound.

DISCUSSION

DR. NELSON PAUL ANDERSON: I rather hesitate to accept the diagnosis of tertiary syphilis. While the serologic reaction is positive, I think these lesions belong in the category of granuloma annulare. The symmetry is against a tertiary lesion. It is true that there is some annular configuration, but there are a number of papules. Further, the eruption does not seem to have the peculiar reddish color one associates with tertiary lesions. It has more the yellowish brown color of granuloma annulare, and I should like to see a specimen for biopsy either from the extensor surface of one forearm or from the back.

DR. H. P. JACOBSON: I agree with the diagnosis of tertiary syphilis. With all due apologies to Dr. Anderson, I cannot subscribe to a diagnosis of granuloma annulare. The entire clinical course, the positive serologic reactions, the morphologic picture—all are consistent with the diagnosis of syphilis.

DR. W. H. GOECKERMAN: I agree with the diagnosis of syphilis, but I should call the lesions a relapsing late secondary eruption, owing to their bilateral and superficial character.

DR. KENDAL FROST: I agree with Dr. Goeckerman's idea. The lesions are characteristic of a type seldom seen, nodular and scar leaving, retaining the symmetry of the early lesions of syphilis.

DR. M. E. OBERMAYER: The transition between the manifestations of the early and those of the late stages of syphilis need not always be sharply distinguished. At times, cutaneous syphilids may exhibit characteristics of both relatively early and late lesions. Such transitional syphilids, of which this patient presents a good example, are solitary or grouped, often orbicular and serpiginous; they do not actually break down but heal, leaving atrophic scars. There is no certain time at which this transitional form of syphilid occurs, for it may appear as early as two years and as late as twenty years after infection.

DR. H. C. L. LINDSAY: Dr. Obermayer has well expressed the fact that the transition from secondary syphilis to tertiary syphilis may show symptoms characteristic of both phases of the disease.

DR. IRVING BANCROFT: These lesions have lasted about three years, which seems a little long for a secondary eruption which has never been treated.

DR. L. F. X. WILHELM: I wish to agree with a diagnosis of late secondary syphilis.

Chronic Radiodermatitis with Ulceration. Presented by DR. KENDAL FROST.

H. A., a 41 year old Negro, a physician, has had the fingers of his left hand in the direct beam of roentgen rays in unmeasured quantities frequently for several years. On Aug. 27, 1942 he accidentally got his hands in extremely hot water for a few seconds. The index and middle fingers of his left hand turned a cherry red and remained so. About October 15 or 16 a door

was slammed on these fingers, and immediately an ulcer appeared, which does not have a tendency to heal. The lesions are extremely painful. On the dorsal surfaces of each of the distal phalanges of the index and middle fingers of the left hand, proximal to the nail folds, is an irregular ulceration. The bases are covered with a somewhat yellowish pellicle. On the outer side of the ulcer on the middle finger is a pea-sized elevated nodule covered with thickened, whitish, macerated epithelium. These lesions are exquisitely painful. For the past two weeks he has been using an ointment containing the pulp of the Aloe vera leaf (veracal ointment), under which the lesion has cleared up somewhat but is not materially changed and is still painful. Previous to that he used 10 per cent naftex (a saponaceous mixture predominantly aliphatic hydrocarbons, with some aromatic and naphthenic hydrocarbons) and 2 per cent ichthaminol in a four base paste for a week without benefit.

DISCUSSION

DR. SAUL ROBINSON: I think this man has a third degree ulcerative radiodermatitis. I think probably the best therapy in this case would be amputation. One must consider the danger of cancer developing and also the severe pain and the time required for healing.

DR. C. R. HALLORAN: I agree with the diagnosis. It seems to me, as far as therapy is concerned, with this man's profession (physician) he needs those fingers, and I should be inclined to wait several months and try conservative measures, such as application of a preparation of Aloe vera, and see whether his fingers might not be spared.

DR. H. P. JACOBSON: The diagnosis in this case is obvious. The patient has a radiation dermatitis associated with radiation necrosis, fairly superficial in character. Dr. Robinson's suggested treatment (amputation of the fingers) reminds me of the Chinese treatment for corns (amputation). I do not believe this method is either indicated or justified in the management of this case. I suggest excision by cautery of the necrotic areas as well as a safe zone of surrounding tissue. Healing would be by granulation and rather slow, but the ultimate results should be satisfactory.

DR. SAMUEL AYRES: I think that would be a good deal too radical in this case. The man never had much trouble until his fingers got in hot water last August; then they were only red until they were slammed in a car door in October. I think traumatic ulceration is what he has, and I should give the Aloe vera ointment a trial for a couple of months, possibly alternating with something else, such as codalltoin ointment (a proprietary ointment containing cod liver oil, allantoin and phenol). There is always time to do some radical work after this.

DR. NELSON PAUL ANDERSON: I think one thing to be considered with this man, who is a physician, is that he is having a tremendous amount of pain. I think a great many persons are started on the road to morphine addiction from roentgen ray ulcerations of great sensitivity, like this patient. He has roentgen ray changes on the dorsum of his ring finger, but there is the secondary element of trauma. I think this condition should be handled conservatively in the matter of conservation of the fingers but radically from the standpoint of excision of the ulcers and stopping the severe pain.

LIEUTENANT COMMANDER LEE MCCARTHY (MC), U.S.N.R. (by invitation): From the experience I have had with radiodermatitis and judging from the amount of destruction of tissue in this case, I should strongly advise amputation of the fingers. Professional men are inclined to delay the issue until the glands are involved. I have known several cases of this type that ended fatally, and I believe it was only because the physician or dentist refused early amputation.

DR. W. H. GOECKERMAN: I should be inclined to be conservative in this case. There is a definite recent traumatic factor. With mild astringent wet dressings and a soothing ointment there should be considerable improvement, which would then allow of better judgment as to just what should be done eventually. I too feel that no matter what improvement one gets, the radiodermatitis is probably of a sufficient degree to necessitate some plastic work later. There could be no objection to the use of an analgesic for the present.

DR. H. C. L. LINDSAY: A preparation which gives relief from pain and suppresses exudation in recent roentgen ray burns is a 10 per cent solution of mild protein silver. The worst roentgen ray burns of the hands that I ever saw were those of some pioneer x-ray operators in Australia. A percentage of those operators in early days had no protection against the rays. I ran the very first x-ray machine in British Columbia. It belonged to the late Dr. R. G. Brett. We had nothing, not even a lead glass bowl, around the tube. Many pioneers in this field lost fingers; some lost hands; some died from keratotic lesions which ultimately developed into cancers. Roentgen ray burns in a way resemble other burns. They vary in depth to a certain extent. If they are not too deep, considerable tissue can be saved sometimes. The outer layers

act as filters, and removal of the outer layers surgically or by paring until one reaches healthy enough tissue and then grafting skin may in certain cases give rapid relief from pain and restore function.

DR. KENDAL FROST: I am grateful for this discussion. I have seen the patient only a few times, and the acuteness of the process led me to think conservative treatment would be best, but he has had such intense pain that I began to feel radical treatment would probably be necessary. I have seen some excellent results from skilful plastic operations in cases of this kind. It might be possible to keep the fingers intact by excision and graft.

Parapsoriasis. Presented by DR. H. C. L. LINDSAY.

J. A., an 80 year old white man, in good health, for two years has had a lichenified, at times slightly scaling rash, tending toward plaque formation; there are ovals of various sizes, which parallel the ribs on the chest and back. These areas are darker in color and slightly indurated. The areas also have been on the hands, where induration was greater, and scaly on the dorsa and bases of the fingers. There is practically no itching, and the general health has been good. Some of the lesions show central scarring of a superficial nature and fine scaling. There are no lesions on the face or in the mouth; none has been on the scalp. The lesions on the backs of his hands were formerly lichenified bluish areas suggestive of scleroderma. This has disappeared, but some disturbance of pigmentation is still present, such as mottled vitiliginous areas, and on the hands there is a line just above the knuckles where the dorsa of the hands are a slight shade darker than the fingers. One patch on the right knee looks somewhat like psoriasis. This patch does itch sporadically, and at times his body itches for a short time. Requestioning on this subject elicits various answers. The patient has had practically no treatment, yet in the course of time the lesions seemed to have faded considerably. He has taken no drugs which tend to cause fixed eruptions. The Wassermann and Kahn reactions of the blood were negative. A biopsy was consistent with a diagnosis of parapsoriasis.

DISCUSSION

DR. C. R. HALLORAN: Clinically, this case impressed me as being one of mycosis fungoides. I think Dr. Lindsay had that in mind, but the section did not support this diagnosis. It falls in that category of cases of either parapsoriasis or mycosis fungoides. There is too much infiltration of some lesions for this case to be one of parapsoriasis. In spite of the histologic picture at present, which fails to support a diagnosis of mycosis fungoides, as time goes on I think you will find that that is what it is.

DR. KENDAL FROST: I think whenever a case of parapsoriasis en plaques is encountered the discussion gets around to the question of mycosis fungoides.

DR. SAUL ROBINSON: In an article by Montgomery and Burkhart (Parapsoriasis: Its Relation to Mycosis Fungoides and Tuberculosis, ARCH. DERMAT. & SYPH. **46**:673 [Nov.] 1942) it is interesting to note that this type is rarely a precursor of mycosis fungoides. The common conception of parapsoriasis en plaques as being really premycotic lesions, I believe, is a misconception.

DR. M. E. OBERMAYER: The diagnosis of parapsoriasis is clinically and histologically well founded. There is yet no evidence of the premycotic stage of mycosis fungoides, a possibility which must be kept in mind.

DR. NELSON PAUL ANDERSON: There was one definitely infiltrated plaque on the back, and I think if Dr. Lindsay would perform a biopsy on a specimen from this lesion he would see changes suggestive of mycosis fungoides.

DR. H. C. L. LINDSAY: Parapsoriasis is difficult to diagnose. In looking over some of the literature, I find that in most cases the disease is not diagnosed for nine months after it is seen by experts. It is diagnosed often by the process of exclusion and by microscopic examination. It either does or does not develop into mycosis fungoides. Time will tell. The slide that Dr. Ford prepared is a good one, and he could not find any signs of mycosis fungoides.

Directory of Dermatologic Societies *

INTERNATIONAL

Tenth International Congress of Dermatology and Syphilology

Oliver S. Ormsby, President, 25 E. Washington St., Chicago
Paul A. O'Leary, Secretary-General, 102-2d Ave. S. W., Rochester, Minn.
Place: New York. Time: Postponed indefinitely.

Pan American Medical Association, Section of Dermatology and Syphilology

Austin W. Cheever, Secretary, 464 Beacon St., Boston.

FOREIGN

British Association of Dermatology and Syphilology (Canadian Branch)

B. Usher, President, 1538 Sherbrooke St. W., Montreal.
Frederick Kalz, Secretary-Treasurer, 1414 Drummond St., Montreal.

Royal Society of Medicine, Section of Dermatology

H. W. Barber, President, 7 Devonshire Pl., London, W. 1, England.
Louis Forman, Secretary, 7 Devonshire Pl., London, W. 1, England.

Sociedad Mexicana de Dermatología

Jorge Millán Gutiérrez, President, Colima No. 208, Mexico, Mexico.
Pedro Daniel Martínez, Secretary, Lucerna No. 48, Mexico, Mexico.

NATIONAL

American Medical Association, Scientific Assembly, Section on Dermatology and Syphilology

Clark W. Finnerud, Chairman, 55 E. Washington St., Chicago.
Nelson P. Anderson, Secretary, 2007 Wilshire Blvd., Los Angeles.

American Academy of Dermatology and Syphilology

Richard S. Weiss, President, 3720 Washington Blvd., St. Louis.
Earl D. Osborne, Secretary, 471 Delaware Ave., Buffalo, N. Y.

American Board of Dermatology and Syphilology

Howard Fox, President, 140 E. 54th St., New York.
C. Guy Lane, Secretary-Treasurer, 416 Marlborough St., Boston.

American Dermatological Association

William H. Guy, President, 500 Penn Ave., Pittsburgh.
Harry R. Foerster, Secretary, 208 E. Wisconsin Ave., Milwaukee.

Society for Investigative Dermatology

Fred D. Weidman, President, 1930 Chestnut St., Philadelphia.
S. W. Becker, Secretary, 8 S. Michigan Ave., Chicago.

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* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

SECTIONAL

Central States Dermatological Association

John E. Rauschkolb, Secretary-Treasurer, 1808 Republic Bldg., Cleveland.
Place: Cleveland. Time: Postponed indefinitely.

Mississippi Valley Dermatological Society

Lawrence C. Goldberg, Secretary-Treasurer, 623 Doctors Bldg., Cincinnati.
Place: Chicago.

New England Dermatological Society

John G. Downing, President, 520 Commonwealth Ave., Boston.
Francis M. Thurmon, Secretary, 520 Commonwealth Ave., Boston.

Northern New Jersey Dermatological Society

John Kiley, President, 94 Park St., Montclair.
Henry Abel, Secretary, 339 Union Ave., Elizabeth.
Place: Academy of Medicine of Northern New Jersey, Newark. Time: Third Tuesday of March, April, October and December.

Southeastern Dermatological Association

Howard Hailey, Chairman, 107 Doctors Bldg., Atlanta, Ga.
A. H. Lancaster, Secretary, 401 Medical Arts Bldg., Knoxville, Tenn.

Southern Medical Association, Section on Dermatology and Syphilology

Dudley C. Smith, Chairman, University of Virginia Hospital, Charlottesville, Va.
J. Lamar Callaway, Secretary, Duke Hospital, Durham, N. C.

STATE

California Medical Association, Scientific Assembly, Dermatology and Syphilology Section

Merlin Trevor-Roper Maynard, Chairman, 241 E. Santa Clara St., San Jose.
Frederick G. Novy, Secretary, 411–13th St., Oakland.

Connecticut State Medical Society, Section on Dermatology

Ralph E. McDonnell, Chairman, 158 Whitney Ave., New Haven.
Louis O'Brasky, Secretary, 1172 Chapel St., New Haven.

Florida Society of Dermatology and Syphilology

Wiley M. Sams, Chairman, 25 S. E. 2d Ave., Miami.
Lauren M. Sompayrac, Secretary, 117 W. Duval St., Jacksonville.

Louisiana Dermatological Society

M. T. Van Studdiford, President, 912 Pere Marquette Bldg., New Orleans.
R. A. Oriol, Secretary-Treasurer, 921 Canal St., New Orleans.

Massachusetts Medical Society, Section on Dermatology and Syphilology

William Boardman, President, 388 Marlborough St., Boston.
J. H. Swartz, Secretary, 371 Commonwealth Ave., Boston.

Medical Society of the State of New York, Section on Dermatology and Syphilology

Rudolph Ruedemann Jr., Chairman, 352 State St., Albany.
Maurice J. Costello, Secretary, 140 E. 54th St., New York.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA, SECTION ON DERMATOLOGY

Paul A. Deckard, Chairman, 814 N. 2d St., Harrisburg.
Bernhard A. Goldmann, Secretary, 500 Penn Ave., Pittsburgh.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Frank Menaugh, Chairman, Ford Hospital, Detroit.
Eugene A. Hand, Secretary, 801 Second National Bank Bldg., Saginaw.

MINNESOTA DERMATOLOGICAL SOCIETY

H. E. Michelson, President, 825 Nicollet Ave., Minneapolis.
F. W. Lynch, Secretary-Treasurer, 317 Lowry Medical Arts Bldg., St. Paul.
Time: First Friday in October, December, February and April.

OKLAHOMA STATE DERMATOLOGICAL SOCIETY

John H. Lamb Jr., President, Medical Arts Bldg., Oklahoma City.
M. M. Wickham, Secretary, 716 W. Symmes St., Norman.

TEXAS DERMATOLOGICAL SOCIETY

Lewis Pipkin, President, 705 E. Houston St., San Antonio.
Duncan O. Poth, Secretary-Treasurer, 414 Navarro St., San Antonio.

LOCAL

BALTIMORE-WASHINGTON DERMATOLOGICAL SOCIETY

H. Hanford Hopkins, Chairman, 1123 St. Paul St., Baltimore.
Reuben Goodman, Secretary-Treasurer, 1746 K St. N. W., Washington, D. C.
Place: Alternate cities. Time: Third Thursday of each month.

BRONX DERMATOLOGICAL SOCIETY

David Bloom, President, 135 E. 50th St., New York.
Henry Silver, Secretary, 290 West End Ave., New York.
Time: Fourth Thursday of each month from October to May, inclusive.

BROOKLYN DERMATOLOGICAL SOCIETY

S. H. Silvers, President, 920 Bushwick Ave., Brooklyn.
M. Estrin, Secretary, 61 Eastern Parkway, Brooklyn.
Time: Third Monday of each month except June, July, August and September.

BUFFALO-ROCHESTER DERMATOLOGICAL SOCIETY

James W. Jordon, President, 471 Delaware Ave., Buffalo.
Frank A. Dolce, Secretary-Treasurer, 468 Delaware Ave., Buffalo.

CENTRAL NEW YORK DERMATOLOGICAL SOCIETY

Leon H. Griggs, President, 1804 State Tower Bldg., Syracuse.
Maxwell C. Snider, Secretary, 106 Oak St., Binghamton.

CHICAGO DERMATOLOGICAL SOCIETY

Michael H. Ebert, President, 25 E. Washington St., Chicago.
Marcus R. Caro, Secretary-Treasurer, 25 E. Washington St., Chicago.

CINCINNATI SOCIETY OF DERMATOLOGY AND SYPHILOLOGY

Harry L. Claasen, President, Provident Bank Bldg., Cincinnati.
Lawrence C. Goldberg, Secretary-Treasurer, 623 Doctors Bldg., Cincinnati.
Place: Cincinnati. Time: First Wednesday of each month, except July, August and September.

Cleveland Dermatological Society

Charles G. La Rocco, President, 669 Rose Bldg., Cleveland.
John E. Rauschkolb, Secretary, 1808 Republic Bldg., Cleveland.

Detroit Dermatological Society

George Van Rhee, President, 10 Peterboro Ave., Detroit.
John H. Besancon, Secretary-Treasurer, 1510 Eaton Tower, Detroit.

Kansas City (Mo.) Dermatological Society

C. C. Dennie, President, 1103 Grand Ave., Kansas City.
Thomas B. Hall, Secretary, 902 Professional Bldg., Kansas City.

Los Angeles Dermatological Society

L. F. X. Wilhelm, President, 727 W. 7th St., Los Angeles.
Arthur Fletcher Hall Jr., Secretary, 710 Wilshire Blvd., Santa Monica.
Time: Second Tuesday of each month, October to May, inclusive.

Manhattan Dermatologic Society

J. Frank Fraser, Chairman, 115 E. 61st St., New York.
Anthony C. Cipollaro, Secretary, 40 E. 61st St., New York.

Montreal Dermatological Society

L. P. Ereaux, President, 1390 Sherbrooke St. W., Montreal, Canada.
Paul Poirier, Secretary, 456 Sherbrooke St. E., Montreal, Canada.

New York Academy of Medicine, Section of Dermatology and Syphilis

Louis Tulipan, Chairman, 50 Park Ave., New York.
Timothy J. Riordan, Secretary, 136 E. 64th St., New York.

New York Dermatological Society

Edward R. Maloney, President, 136 E. 64th St., New York.
J. Gardner Hopkins, Secretary-Treasurer, 630 W. 168th St., New York.

Omaha Dermatological Society

Donald J. Wilson, President, 1113 Medical Arts Bldg., Omaha.
Leonard J. Owen, Secretary-Treasurer, 954 Stuart Bldg., Lincoln, Neb.

Philadelphia Dermatological Society

Carroll S. Wright, Chairman, 1402 Spruce St., Philadelphia.
Carmen C. Thomas, Secretary, 1930 Chestnut St., Philadelphia.
Time: Third Friday of September, November, January, March and May.

Pittsburgh Dermatological Society

M. F. Pettler, President, 1319–8th Ave., Beaver Falls, Pa.
Frederick Amshel, Secretary, 3401 Fifth Ave., Pittsburgh.
Time: Third Thursday of every month except July and August.

St. Louis Dermatological Society

Norman Tobias, President, Missouri Theatre Bldg., St. Louis.
Joseph Grindon Jr., Secretary-Treasurer, 323 Lister Bldg., St. Louis.
Place: Barnard Free Skin and Cancer Hospital. Time: 2 p. m., second Wednesday of each month.

San Francisco Dermatological Society

Arne E. Ingels, President, 490 Post St., San Francisco.
Ervin Epstein, Secretary-Treasurer, 1904 Franklin St., Oakland, Calif.
Time: Third Friday of February, April, September and November.

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